A STUDY AND ANALYSIS OF PANNICULITIDES

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CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY AND ANALYSIS OF PANNICULITIDES"** submitted by **Dr.R.Madhavan** to The Tamil Nadu Dr. M. G. R. Medical University, Chennai is in partial fulfillment of the requirement for the award of M.D., (DERMATO VENEREO LEPROLOGY) and is a bonafide research work carried out by him under direct supervision and guidance.

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DECLARATION

I, **Dr. R.Madhavan** solemnly declare that I carried out this work on **"A STUDY AND ANALYSIS OF PANNICULITIDES"** at Department of Dermatology, Government Rajaji Hospital during the period of April 2008 – October 2009.

I also declare this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any university, board either in India or abroad.

This is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulation for M.D., (D.V.L) Degree examination.

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CONTENTS

INTRODUCTION

The panniculitides comprise a heterogeneous group of inflammatory diseases involving the subcutaneous fat. Panniculitis are classified based on histopathological parameters. Panniculitides have been traditionally divided into septal panniculitis, lobular panniculitis, mixed panniculitis and panniculitis with vasculitis.

Working classification[5] divides panniculitis into mostly septal[1] or mostly lobular[2] panniculitis, according to the location in which the inflammatory infiltrate is more abundant. They are further divided based on the presence or absence of vasculitis and nature of inflammatory infiltrate.

From a clinical standpoint, many forms of panniculitis of diverse etiology closely resemble one another, presenting as tender erythematous subcutaneous nodules. Some panniculitides can be a manifestation of different disease processes, and even if the type of panniculitis is correctly identified, this is only the first step in the series of clinical and laboratory investigations required to determine the underlying causes. From a p athologic standpoint, the subcutaneous fat responds to a variety of insults in a limited number of ways, and therefore, the histopathological difference among various forms of panniculitis may be subtle.

Histopathologically there may be overlap between the various forms of panniculitis. Most panniculitis are persistent, lasting for weeks or months. For diagnostic purposes, the biopsy should be taken from an active earlier lesions and serial biopsy may be required in certain conditions

Most of the studies on panniculitides are from the western countries. Studies on this uncommon group of diseases in Indian literature are sparse. We have conducted this study to find out the different patterns and common types of panniculitis in our population.

REVIEW OF LITERATURE

The panniculitides comprise a heterogeneous group of inflammatory diseases involving the subcutaneous fat. These disorders have been considered diagnostically challenging, for the dermatologist. Study of panniculitides has several pitfalls. First of all, with few exceptions, most panniculitis exhibits same (monotonous) clinical features, namely erythematous nodules generally located on the lower limbs. Second, superficial and inadequate biopsy specimens are often obtained for histopathological study. A third difficulty for the specific diagnosis of panniculitis results from the evolutionary nature of the lesion, because the composition and the distribution of inflammatory cells change within the course of few days.

Despite all these difficulties, a specific diagnosis can usually be made with appropriate biopsy and an adequate clinicopathological correlation aided by various investigational profiles.

Anatomy and Histology of Subcutaneous tissue[5]:

The basic unit of the subcutaneous fat is the primary micro lobule, which measures approximately 1 mm in diameter and is composed of microscopic collection of adipocytes or lipocytes.

Primary microlobules aggregate to form secondary lobule that measure approximately 1 cm in diameter and are surrounded by thin septa

of connective tissue. The septa provide stability to the subcutaneous tissue by compartmentalization.

The individual adipocytes are large up to 100 um diameter, with H & E stain appearing as empty cell with ‘signet ring’ morphology. This is because the lipid content dissolves in routinely processed specimens and the flat spindle shaped nucleus is displaced to the periphery of the cells by a single, large intracytoplasmic vacuole.

The septa that divide the subcutaneous fat into lobules are composed of collagen and reticulin fibers that are extensions of the dermis. These septa house the blood and lymphatic vessels and the nerves. Arteries and veins of the subcutis run along the septa. Each individual secondary lobule is supplied by a small muscular artery branching from the septa to form arterioles that supply every individual primary microlobule. The arteriole branches to form capillaries into the microlobule, and a capillary network surround each individual adipocyte. Post capillary venules meet in veins, that run along the septa. In each microlobule, the arteriole occupies a central position, whereas the venule runs along the periphery[5]. As a consequence, interference with the arterial supply results in diffuse changes within the lobule (mostly lobular panniculitis) where as the venous disorders are manifested by alteration is the septal and paraseptal areas (mostly septal panniculitis).

The blood supply to each microlobule is terminal. This peculiar structure of the blood supply in subcutaneous fat explains why large vessel vasculitis involving the septal vessels is usually accompanied by little inflammation of the fat lobules, where as when the vasculitis involves the small blood vessels, there is extensive necrosis of adipocytes with centrilobular infarct and dense inflammatory infiltrate within the lobule.

Classification of Panniculitis [1-4]

Panniculitis are broadly classified into septal panniculitis, lobular panniculitis, mixed panniculitis and panniculitis with vasculitis.

A. Septal Panniculitis

1. Erythema nodosum
2. Erythema nodosum migrans
3. Eosinophilic panniculitis (this condition may overlap with lobular or mixed panniculitis)

B) Lobular Panniculitis

1. Relapsing febrile nodular panniculitis (Weber Christian syndrome)
2. Idiopathic nodular panniculitis
3. Lipoatrophic panniculitis (formerly Rothman – Makai syndrome)
4. Panniculitis associated with crystal deposition – Sclerema neonatorum, subcutaneous fat necrosis of new born, gout or factitial panniculitis, post steroid panniculitis.

5. Enzymatic (Pancreatic) Panniculitis
6. Alpha-1 antitrypsin deficiency panniculitis
7. Fat necrosis – cold injury, nodular cystic fat necrosis, lipomembranous
8. Lymphomatous panniculitis
9. Cytophagic histiocytic panniculitis.

C) Mixed Panniculitis:

1. Lupus erythematous profundus
2. Scleroderma (Fasciitis with eosinophilia)
3. Connective tissue panniculitis (overlaps with lipoatrophic panniculitis)
4. Subcutaneous sarcoidosis
5. Subcutaneous granuloma annulare
6. Necrobiosis lipoidica
7. Infective panniculitis (e.g. opportunistic bacterial or fungal infections)
8. Physical and factitious panniculitis (e.g. sclerosing lipogranuloma, oil granuloma)
9. Sclerosing panniculitis (lipodermatosclerosis)
10. Fasciitis – Panniculitis syndrome

D – Panniculitis with vasculitis

Based on the size of the vessel involved

1. Small vessel vasculitis - Leukocytoclastic vasculitis
2. Large vessel vasculitis - Polyarteritis nodosa
   Nodular vasculitis
   Thrombophlebitis

3. Neutrophilic panniculitis

4. Oedematous scarring vasculitic panniculitis

Subsequent working classification[5] divides panniculitis into predominantly septal or predominantly lobular panniculitis[1,2]. Further classification can be based on the presence or absence of vasculitis and the type of inflammatory infiltrate observed. A classification based on the sequence of inflammatory cell infiltrate has been proposed by White WL et al[6].

Working classification [1, 2, 5]

Classification of the Panniculitides

Mostly septal panniculitides

With vasculitis

Veins: Superficial thrombophlebitis

Arteries: Cutaneous polyarteritis nodosa

No vasculitis

Lymphocytes and plasma cells mostly

- With granulomatous infiltrate in septa: Necrobiosis lipoidica
- No granulomatous infiltrate in septa : Deep morphoea

Histiocytes mostly: granulomatous infiltrate

- With mucin in center of palisaded granulomas: Subcutaneous granuloma annulare
- With fibrin in center of palisaded granulomas: Rheumatoid nodule
- With large areas of degenerated collagen, foamy histiocytes, and cholesterol clefts: Necrobiotic xanthogranuloma
- Without mucin, fibrin, or degeneration of collagen, but with radial granulomas in septa: Erythema nodosum

Mostly lobular panniculitides

With vasculitis

Small vessels

Venules: Erythema nodosum leprosum

Lucio phenomenon

Large vessels

Arteries and veins: Erythema induratum of Bazin

No vasculitis

Few or no inflammatory cells

- Necrosis at the center of the lobule: Sclerosing panniculitis.

- With vascular calcification: Calciphylaxis
- With needle-shaped crystals in adipocytes: Sclerema neonatorum

Lymphocytes predominant:

- With superficial and deep perivascular dermal infiltrate: Cold panniculitis
- With lymphoid follicles, plasma cells, and nuclear dust of lymphocytes: Lupus panniculitis.

Neutrophils predominant

- Extensive fat necrosis with saponification of adipocytes: Pancreatic panniculitis.
- With neutrophils between collagen bundles of deep reticular dermis: $Alpha_1$ – antitrypsin deficiency panniculitis
- With bacteria, fungi, or protozoa: Infective panniculitis
- With foreign bodies: Factitial panniculitis

Histiocytes predominant (granulomatous)

- No crystals in adipocytes

  Subcutaneous sarcoidosis

  Traumatic panniculitis

  Lipomembranous fat necrosis

  Lipodystrophy and lipoatrophy

- With crystals in histiocytes or adipocytes

  Subcutaneous fat necrosis of the newborn

  Poststeroid panniculitis

- With cytophagic histiocytes: cytophagic histiocytic panniculitis

  Panniculitis-like subcutaneous lymphomas

- With sclerosis of the septa: Sclerosing postirradiation panniculitis

Septal Panniculitis without vasculitis.

Erythema nodosum[1, 6, 7-15]

Erythema nodosum is the most frequent[1] clinicopathological variant of panniculitis. Most cases appear between 2nd and 4th decades of the life, with the peak of incidence between 20 and 30 years of age. Several studies have demonstrated that Erythema nodosum occurs three to six times more frequently in women than in men[7]. Most cases of erythema nodosum occur within the first half of the year probably, because of the more frequent incidence of streptococcal infections in this period of the year. The typical eruption is quite characteristic and consists of a sudden onset of symmetric, tender, erythematous, warm nodules and raised plaque usually located on the shins, ankles and knees.

The size of the nodules ranges from 1-5 cm or more in diameter. The color of the nodules finally exhibits a yellow or greenish appearance often taking on the look of a deep bruise (erythema contusiformis) Ulceration is never seen in erythema nodosum and the nodules heal without atrophy or scarring. Usually, acute bouts of erythema nodosum are associated with a fever of 38^{o} C to 39^{o} C, fatigue, malaise, arthralgia, headache, abdominal pain, vomiting, cough or diarrhoea. Episcleral lesions and phlyctenular conjunctivitis may also accompany the cutaneous lesion. Eruption last for 3-6 weeks [1, 6].

Variants of erythema nodosum.

Three variants of erythema nodosum have been described.

Erythema nodosum migrans[14]

Erythema nodosum migrans is clinically and histopathologically different from classical erythema nodosum. The lesions are unilateral, less tender and they consist of erythematous plaques that extend peripherally and heal at the centre and are preferentially located on the lateral than on the anterior side of the leg. Often there is a protracted clinical source with patients exhibiting few systemic symptoms.

Subacute nodular migratory panniculitis of vilanoa and pinol[15]

Subacute nodular migratory panniculitis although thought to have been a distinct subset of erythema nodosum, is now considered to be same as erythema nodosum migrans.

Chronic erythema nodosum[9]

It was initially used to describe erythema nodosum migrans or subacute nodular migratory panniculitis. Recently chronic erythema nodosum and erythema nodosum migrans are considered as two different entities[8]. It is a common nodular inflammatory eruption usually affecting the legs of adult women, distinguished from erythema nodosum by it is prolonged unpredictable course which may continue for years[9].

Prevalence of Erythema nodosum:

Frequency of erythema nodosum in a study done on panniculitis over a four year period in India by Handa et al[10] was 79.3%.There are a variety of etiological factors that lead to erythema nodosum, these includes infections, drugs, malignancies and a wide group of miscellaneous conditions .Majority of cases in the Indian study by Handa et al[10] were idiopathic. Streptococcal infection and sarcoidosis were the prominent causes among patients with erythema nodosum in the west. [11, 12]

Histopathologically, erythema nodosum is the stereotypical example of a mostly septal panniculitis with no vasculitis. Superficial and deep

perivascular inflammatory infiltrate predominantly composed of lymphocytes and histiocytes, is also seen in the overlying dermis. In early lesion, oedema, hemorrhage and neutrophils are responsible for the septal thickening, whereas fibrosis, periseptal granulation tissue, lymphocytes, histiocytes and multinucleated giant cells are the main findings in late stage. A histopathologic hall mark of erythema nodosum is the presence of the so called “Miescher’s radial granuloma[13] which consist of small well defined nodular aggregations of small histiocytes around small blood vessels or slit like spaces. The fat lobules can become progressively replaced and effaced by widening septa which can completely obliterate the lobules. Macrophage granulomas formed in the septa, without lipid deposition, are more frequent when late lesions are compared to early ones.

Histopathologically, lesions of erythema nodosum migrans showed marked thickening of septa of subcutaneous fat with an abundant number of granulomas containing frequent multinucleate giant cells, granulations tissue and conspicuous proliferation of capillaries at the separation between septa and fat lobules. There is no phlebitis.

Lesions of chronic erythema nodosum showed lesser degree of thickening of the septa and less inflammatory infiltrate but phlebitis and extravasated erythrocytes were prominent and characteristic findings.

Eosinophilic Panniculitis[16]

Characterized by a prominent infiltration of subcutaneous fat with eosinophils, principally infiltrating the septa but can involve the lobules. The current view of eosinophilic panniculitis is that it is not a disease entity, but it can be principally considered as a reactive process, often associated with a systemic condition. More common cause of eosinophilic panniculitis include arthropod bites, erythema nodosum, vasculitis, well's syndrome, eosinophilic fasciitis, hypereosinophilic syndrome, and eosinophilic leukemia.

Histopathologically, there is a mixed cell inflammatory infiltrate of lymphocytes, macrophages, and numerous eosinophils, involving both septa and lobules of subcutaneous tissue.

Subcutaneous Morphoea[17-19]

The term subcutaneous morphoea and morphoea profunda are often used interchangeably, although morphoea profunda has been proposed as the appropriate diagnosis when both subcutaneous fat and fascia are involved and subcutaneous morphoea for cases with involvement of fat only[17]. The term Morphoea profundus was first suggested by Whittaker et al[18] in 1989, to describe solitary fibrotic plaque on shoulder, back, neck or paraspinal area. The overlying skin may be pigmented or hypopigmented.

Histopathologically, a distinctive septal panniculitis without vasculitis forming fenestrated pattern by widened septa and shrunken lobules. Predominant lymphoplasmocytic infiltrate especially at dermal-subcutaneous interface with unique dermal and subcutaneous sclerosis.

Eosinophilic fasciitis (Shulman's syndrome) [20-21]

A group of disorder characterized by sudden onset of symmetric induration of the skin and subcutaneous tissue. Recognized as a distinct variant of morphoea because of its sudden onset, its usual limitation to the structures underlying the skin and its tendency to resolve spontaneously[21].

Histopathologically, the earliest changes occur in the interlobular septa of subcutis and deep fascia. There is oedema and inflammatory infiltrate of lymphocytes, histiocytes, plasma cells and prominent eosinophils. Eventually there is a thickening of deep fascia and septa of subcutis with fibrosis and hyalinization of collagen. This process extends into deep dermis where there is atrophy of appendages with sclerosis of lower dermis.

Subcutaneous nodules of juvenile rheumatoid arthritis[22]

Subcutaneous nodules occur in still's disease. These nodules resemble that seen in the nodules of rheumatic fever rather than in those of rheumatoid arthritis. In most case the nodules are indistinguishable from

those of rheumatic fever, showing absence of necrosis and palisading and presence of oedema, well marked vascular islands and a fibrinoid lattice.

Lobular Panniculitis without vasculitis.

Relapsing febrile nodular panniculitis[23-24]

(Weber Christian syndrome)

It is a febrile disease, characterized by the recurrent formation of single or multiple crops of tender inflammatory subcutaneous nodules. Histopathologically the lesions usually showed mostly lobular panniculitis without vasculitis. The term nodular panniculitis was preferred for this entity [24]. Because of the variation in clinical presentation, it should not be considered as a distinct clinical entity at present. On further investigations, many cases of nodular panniculitis can be subsequently reclassified depending on their cause; for example, pancreatic panniculitis, Alpha-1 antitrypsin deficiency or cytophagic panniculitis.

Idiopathic nodular panniculitis[25]

It is characterized by recurrent crops of nodular panniculitis. Histopathology showing lobular Panniculitis without vasculitis. 50% of cases are idiopathic. Majority of cases are young female adults. Presenting with dull red tender subcutaneous nodules usually 1-2 cm in diameter, tend to be maximal on the lower limbs.

Lipoatrophic Panniculitis[26]

Lipoatrophic panniculitis refers specifically to loss of subcutaneous fat due to previous inflammatory process involving the subcutis. It is a residual process of severe inflammatory conditions involving the subcutaneous fat lobules.

Histopathologically, it is a lobular panniculitis which can be divided into two types namely involutionary and inflammatory. Involutional lipoatrophy is associated with a single localized area of lipoatrophy and absence of serological abnormalities. Inflammatory lipoatrophy is associated with multiple areas of lipoatrophy and presence of serological abnormalities consistent with connective tissue disorder.

HIV-Associated Lipodystrophy[53, 54]

This is particularly seen in HIV patients on long term protease inhibitors and patients on nucleoside reverse transcriptase inhibitors, specifically stavudine[53]. It usually starts in the first 6 to 12 months after beginning therapy. The patient may presents with three forms

1) generalized or localized lipoatrophy of face, extremities and buttock,

2) lipohypertrophy with generalized or local fat deposition involving the abdomen, breast, and supraclavicular, and dorsocervical regions(buffalo hump) and 3) a mixed pattern with central adiposity and peripheral lipoatrophy [53]

Histopathologically, in fully developed lesions of lipodystrophy, there is an absence of subcutaneous fat with deposition of new collagen. Recently, two histopathological variants[54] of lipodystrophy have been proposed .In the first type there are prominent involutional changes in fat lobule, with small adipocytes with intervening hyaline or myxoid connective tissue and proliferation of blood vessels. The second type is the inflammatory type, since lymphocytes, foamy histiocytes, and plasma cells appearing with in the small fat lobules with normal appearing adipocytes and vasculature.

Panniculitis associated with crystal deposition

Sclerema Neonatorum[27]

It is a very rare disorder that almost always appears during first week of life, described in premature and small for date neonates. Characterized by hardening of the skin that gets bound down to the underlying muscle and bone, hindering respiration and feeding and is associated with congenital anomalies, cyanosis, respiratory illnesses and sepsis. Histology of the skin biopsy shows thickening of the trabeculae supporting the subcutaneous adipose tissue and a sparse inflammatory infiltrate of lymphocytes, histiocytes and multinucleate giant cells. The adipocytes contain needle shaped clefts in radial array. Sclerema neonatorum has a high case fatality rate

Subcutaneous fat necrosis of the newborn[28, 29]

Transient rare disorder of neonates characterized by focal areas of fat necrosis cause nodular skin lesions. Generally occurs in full term or post term infants of normal birth weight during first few days of life. The lesions consist of bluish red plaques and nodules that has a predilection for the thighs, buttocks, cheeks, back and arms. It may occasionally be associated with symptomatic hypercalcaemia for up to 3 months after birth. The condition has been found to be associated with hypothermia, obstetric trauma, maternal diabetes and maternal preeclampsia.

Histopathologically, there is a lobular panniculitis with a mixed inflammatory infiltrate including giant cells. Adipocytes and giant cells contain needle shaped clefts, which are never present in cold panniculitis[28,29]

Post - steroid Panniculitis[2]

Rare variant of lobular panniculitis that has been reported in children receiving high doses of systemic corticosteroids after the therapy is discontinued. The lesions appear as small painful nodules, which spread in the cheeks, arms and trunk.

Histopathologically, there is a lobular panniculitis with histiocytes, giant cells and lymphocytes. There are needle shaped clefts within adipocytes and multinucleate giant cells.

Calcifying Panniculitis with renal failure [30-33]

In patients with chronic renal failure and secondary hyperparathyroidism, high parathyroid hormone levels lead to the process of calciphylaxis and metastatic calcification. The calcification is principally present within smaller and medium sized arteries. Younger age group and long term haemodialysis were considered as risk factors[33]. It begins with erythematous tender nodules or plaques that progress to violaceous livedo like areas usually located on the thigh, abdomen or buttock. These lesions tend to increase rapidly in size, progressing to large necrotic ulcers.

Most of these patients have elevated serum calcium and phosphorous due to secondary hyperparathyroidism associated with renal failure[31]. In the absence of renal pathology it has been associated with primary hyperparathyroidism, hypercalcaemia of malignancy[32]and in AIDS patients with renal failure[33].

Histopathological features of cutaneous lesion of calciphylaxis are characteristic. They consist of calcium deposition in the walls of small to medium sized blood vessels of reticular dermis and subcutaneous fat. They are associated with ischemic lobular fat necrosis, intralobular calcifications and inflammatory infiltrate of lymphocytes, neutrophils and foamy histiocytes.

Gout[34]

Rarely lobular panniculitis can be caused by deposition of crystals secondary to hyperuricaemia. These urate crystals are inflammatory, fine needle shaped and tendency to form sheaves.

Enzymatic Panniculitis: (Pancreatic panniculitis) [35-39]

Panniculitis developing in patients with pancreatic disease was first described by chiari[35] in 1883. It occurs in 2-3% of all patients with pancreatic disorders. The most common underlying pancreatic disorders associated with pancreatic panniculitis are acute[36] and chronic pancreatitis, which usually results from alcohol abuse, trauma or cholelithiasis but, it has also been described as a complication of pancreatic carcinoma. Skin eruption without abdominal symptoms may be the presenting symptoms in patients with pancreatic fat necrosis.

Clinically, pancreatic panniculitis presents with ill defined, tender, oedematous, erythematous or red brown nodules that may spontaneously ulcerate and drain an oily brown sterile and viscous substance that results from liquefaction necrosis of adipocytes. These lesions are usually located in the distal parts of lower extremities around the ankles and knees. Monoarticular or oligoarticular arthritic symptoms due to focal necrosis of the periarticular fat reported in 56% of the patients in the series reported by Dahl and co workers[37]

Histopathologically, predominantly lobular panniculitis without vasculitis. Biopsy specimens from fully developed lesions of pancreatic panniculitis show a characteristic coagulative necrosis of the adipocytes ,which leads to 'ghost adipocytes'[38]. Ghost adipocytes are cells that lost their nucleus and have a thick shadowy wall with fine basophilic granular or homogenous material within the cytoplasm as a result of calcium deposits. Dystrophic calcifications are seen, which are due to the saponification of fat secondary to the hydrolytic action of pancreatic enzymes on subcutaneous fat, followed by calcium deposit. Inflammatory infiltrate is more of granulamatous, containing foamy histiocytes, multinucleate giant cells, and hemosiderin deposition. Fibrosis and lipodystrophy are seen in final stages. In the very early stage of pancreatic panniculitis a septal pattern has been described with neutrophilic infiltrate may be found at the periphery of fat necrosis areas.

Alpha -1 Antitrypsin Deficiency Panniculitis (A1AT) [40-41]

Rubinstein and Colleagues described[40] the first two cases of A1AT deficiency related panniculitis. Subcutaneous fat necrosis due to A1AT deficiency most commonly occurs during adult life. Trauma and surgical debridements or even cryosurgery are frequent precipitating factors of panniculitis of A1AT deficiency.

The lesions of A1AT deficiency panniculitis occurs predominantly on the trunk and proximal extremities as erythematous nodules, plaque and frequently develop ulcer that drain an oily material[41]. Healing is accompanied by atrophic scars. Chronicity and recurrences are common.

Histopathology of cutaneous lesion of A1AT deficiency Panniculitis show severe necrosis of fat lobules. There is splaying of neutrophils between collagen bundles of reticular dermis.

Fat necrosis:

These include cold panniculitis, nodular cystic fat necrosis and lipomembranous fat necrosis.

Cold Panniculitis[42, 43]

It is a form of localized panniculitis that results from cold injury to subcutaneous fat. Infants are more susceptible than adults[42]. Two types: 1.Haxthausen's disease or Popsicle panniculitis 2.Equestrian cold panniculitis. They manifest clinically by indurated erythematous plaque with ill defined margins that occurs 48 to 72 hrs after exposure to cold. Cold Panniculitis was also reported during the cold months in the thigh or buttock of woman who ride horses wearing tight trousers[43]

Histopathologically, cold panniculitis shows mostly lobular panniculitis with an inflammatory infiltrate of lymphocytes and histiocytes in the fat lobules with marked oedema in the papillary dermis as well as

superficial and deep perivascular infiltrate in the dermis that extends to the subcutaneous septa.

Nodular cystic fat necrosis[44]

Form of encapsulated subcutaneous fat necrosis that presents as solitary or multiple subcutaneous nodules. The lesions are painless occur often in the lower leg of healthy adolescent boys or middle aged women. The size of the lesions varying from 2 to 35 mm in diameter. History of preceding trauma is present in 30% of cases.

Histopathology consists of lesions showing encapsulated fat necrosis with preservation of outlines of non nucleated adipocytes with minimal inflammatory changes. Dystrophic calcifications may be seen in older lesions.

Lipomembranous or Membranocystic Panniculitis [45-47]

Primary lipomembranous panniculitis is a specific and hereditary form of lobular panniculitis associated with anomalies of long bones and neuropsychiatric manifestation. Secondary lipomembranous panniculitis is mostly seen in late stage lesions of Sclerosing panniculitis[45] but also has been described in various other forms of panniculitis.

Histopathology of lesion of primary lipomembranous panniculitis[46] shows multiple microcysts lined by membranes and light brown granules in the histiocytes. Secondary lipomembranous panniculitis[47] changes were

noted in morphoea profunda, lupus panniculitis, and factitial ulcer. Microcysts were formed by the coalescence of the destroyed fat cells and were lined by amorphous, eosinophilic material. Some of the linings had a crenelated appearance.

Cytophagic Histiocytic Panniculitis[48-50]

Cytophagic histiocytic panniculitis (CHP) was first described in 1980 by Winkelmann[48] as a chronic histiocytic disease of subcutaneous adipose tissue, characterized clinically by tender erythematous nodules, recurrent high fever, malaise, Jaundice, organomegaly, serosal effusions, pancytopenia, hepatic dysfunction and coagulation abnormalities[48]. CHP may occur either isolated or as a part of cutaneous manifestation of haemophagocyctic syndrome (HPS). It is rare and often fatal form of panniculitis with multisystem involvement but it can also present in a benign form involving only subcutaneous tissue.

Histologically, it begins as a type of regional histiocytosis that primarily involves subcutis[49]. The histiocytes are actively cytophagic, so that they become stuffed with blood cells, red cells, nuclear fragments and platelets thus giving them a characteristic **"bean bag appearance"**. It is a lobular panniculitis with areas of fat necrosis, together with massive hyaline necrosis, oedema and hemorrhage.

In a study by Marzano et al[50], it was thought that cytophagic histiocytic panniculitis and subcutaneous panniculitis like T cell lymphoma (SPLT) may span a clinicopathological spectrum in which there is a natural progression from cytophagic histiocytic panniculitis to subcutaneous panniculitis like T cell lymphoma[50].

Subcutaneous Panniculitis like T cell lymphoma [51,52]

This is a term recognized by international lymphoma study group in 1994 and is categorized as a subtype of peripheral T cell lymphoma corresponding to pleomorphic medium and large cell lymphoma by the criteria of updated Kiel classification[51] . Perniciaro et al[52] suggested that SPTL could pursue two distinct courses - protracted course and rapidly progressive course[51].Sites of cutaneous involvement varied with extremities most commonly affected followed by trunk. Nearly all patients presented with multiple tan to red non tender subcutaneous nodules or plaques ranging from 0.5 cm to 13 cm in diameter.

Histopathologically, lobular panniculitis are seen with neoplastic lymphocytes and marked atypical, large hyperchromatic nuclei.

Mixed Panniculitis without vasculitis

Lupus erythematosus panniculitis [55-60]

Lupus erythematosus panniculitis is a clinical variant of lupus erythematosus, which may be the unique manifestation or appear before or

after the clinical onset of DLE or systemic lupus erythematosus (SLE) [56]. It may affect both gender, but is more frequent in women. The age of presentation usually ranges between 30-60 yrs. Median age was 41 years old. Affect slightly younger age group in Asian Population[55]. It can be associated with neonatal lupus[58].

It is a chronic recurrent panniculitis that appears in 2% of patients with cutaneous lupus erythematosus[55]. Lupus erythematosus panniculitis consists of tender deep subcutaneous nodules or plaques that sometime arise in crops. The usually involved sites are proximal extremities, particularly the lateral aspects of the arms and shoulders, buttocks, trunk, breast, face and scalp. Lupus erythematosus panniculitis involving legs is unusual, this feature distinguish it from other panniculitis. Erythema is a common clinical feature in the overlying skin and if clinical data of DLE are present skin surface may show scaling, follicular plugging, atrophy, dyspigmentation, telangiectasias or ulceration[59]. Ulceration occurs in 28% of the patients.

The frequency of association of Lupus erythematosus panniculitis to DLE is variable ranging between 21-60%. Lupus erythematosus panniculitis occurs only in 2 to 5% of the patients with SLE.

Histopathologically, in more than half of the cases, there are epidermal and dermal changes of DLE. It is a mixed panniculitis involving

both lobules and septa. Peters and Su[60] have proposed some histopathological criteria for the diagnosis of Lupus erythematosus panniculitis. Major histological criteria needed for the diagnosis of lupus erythematosus panniculitis include 1) hyaline fat necrosis, 2) lymphocytic aggregates and lymphoid follicle formation 3) periseptal or lobular lymphocytic panniculitis and 4) calcification. Minor criteria include 1)overlying changes of discoid LE 2) hyalinization of the subepidermal zone 3) mucin deposition 4) lymphocytic vascular inflammation 5) infiltration of plasma cells and eosinophils 6) histiocytes and small granulomas

Panniculitis in Dermatomyositis: [61-63]

Panniculitis associated with dermatomyositis is extremely rare. Clinically it presents as subcutaneous, indurated, painful nodular lesions over arms, buttock, thigh and abdomen[61].It has been reported in four children with dermatomyositis[62].

Histopathological findings in lesions of panniculitis associated with dermatomyositis were similar to those of lupus panniculitis. The commonest histopathological finding is lobular panniculitis with lymphocytes, histiocytes and epitheloid cells. Membranocystic changes have been reported[63].

Connective Tissue Panniculitis: [64-66]

It was described by Winkelmann[64] and Padilha – Goncalves in 1980. It is a unique panniculitis with transient ANA and no definite pathological or clinical relationship to other connective tissue disorders. Connective tissue panniculitis and focal lipoatrophy may be forms of immunoreactive panniculitis or connective tissue disease (or both) in which typical microscopic, serologic, or other events related to classic connective tissue disease have not occurred. Lesions appeared on the shoulders, upper arms and more rarely on cheeks, neck or legs. The lesions were tender subcutaneous nodules and plaques with slight erythema or no change in skin color[64]. At times the lesions healed with subcutaneous atrophy.

Histopathologically, it shows a lobular or mixed panniculitis composed of lymphocytes and histiocytes with areas of caseous necrosis of fat tissue which can lead to prominent lipoatrophy[65]. Lymphoid follicle and hyaline fat necrosis as seen in lupus panniculitis are not present[66].

Subcutaneous Sarcoidosis [67-69]

Subcutaneous sarcoidosis is a rare cutaneous expression of systemic sarcoidosis, reported to occur in 1.4 to 1.6% of patients with systemic Sarcoidosis[67].Because subcutaneous Sarcoidosis can be asymptomatic and goes unrecognized.

It occurs most often in fifth and sixth decades, presenting as multiple hardly indurated subcutaneous nodules without changes in the overlying epidermis. Usually located symmetrically on the extremities and may persist for much longer periods of time.

In the largest series by Ahmed and Harstad[68], a systemic disease component was recognized in 16 to 20 patients (80%) at the time subcutaneous sarcoidosis was diagnosed, mainly with bilateral hilar adenopathy. Subcutaneous sarcoidosis usually appears at the beginning of sarcoidosis. Demonstration of sarcoidal granulomas in subcutaneous tissue may be useful for establishing the diagnosis of systemic sarcoidosis and avoiding more aggressive diagnostic procedures.

Histopathologically, subcutaneous sarcoidosis shows an inflammatory infiltrate composed of noncaseating granulomas involving fat lobules[69],that are usually sharply demarcated in the dermohypodermic junction. The inflammatory infiltrate is predominantly lobular with minimal or no septal involvement, and it appears as lobular panniculitis.

Subcutaneous Granuloma annulare[70]

It occurs predominantly in children. Lesions are nodular and mostly occur on the scalp and legs, particularly in a pretibial location[70.] In 25% of cases, lesions of subcutaneous granuloma annulare coexist with classical findings of granuloma annulare in the dermis.

The histopathological features consist of areas of necrobiosis with peripheral palisading granulomas involving the septa of subcutis.

Necrobiosis lipoidica[71]

Necrobiosis lipoidica is a disorder of collagen degeneration with a granulamatous response, thickening of blood vessel walls and fat deposition. The exact cause of Necrobiosis lipoidica is unknown, but the leading theory of necrobiosis lipoidica has focused on diabetic microangiopathy, described in about 0.3% of diabetic patients. Typical lesions occur on the pretibial skin and begin as a firm, dull red papule or plaque, which enlarges radically to become a yellowish atrophic plaque with an erythematous edge. The surface is often glazed in appearance, and telangiectatic vessels may be prominent. Lesions are usually asymptomatic. Anesthesia and hypohidrosis are features of the affected skin. In most cases the lesions are bilateral.

Histopathologically, necrobiosis lipoidica presents with interstitial and palisaded granulomas that involve the dermis and septa of subcutaneous tissue. The important findings on histopathology are thickening of blood vessel wall and endothelial cell swelling found in the middle to deep dermis.

Infective Panniculitis [72-77]

Any kind of microorganism can cause inflammation of subcutaneous fat. They are mainly lobular panniculitis without vasculitis but can present as a mixed panniculitis. Clinical presentation of infective panniculitis is usually non specific, so its diagnosis requires microbiological and histological studies.

Bacterial Panniculitis caused by streptococcus pyogenes, staphyloccous aureus[72], pseudomonas spp, klebsiella, Nocardia spp and brucella. Most of the cases have been described in immunosuppressive patients.

Most of the cases of mycobacterial panniculitis reported in the literature were caused by nontuberculous mycobacteria[73], most were caused by rapidly growing mycobacteria, such as mycobacterium chelonae, mycobacterium fortuitum.

Mycobacterium ulcerans causes a well defined clinicopathological entity known as buruli ulcer, which involves predominantly the subcutaneous fat[74].

Fungal panniculitis by any type of subcutaneous mycosis, with most common being sporotrichosis by sporothrix schenckii, eumycetoma caused by madurella mycetomatis, chromoblastomycosis caused by pigmented fungi phialophora verrucosa and cladophialophora carrionii. Rare infection

include phaeohypomycosis, lobomycosis, rhinosporidiosis and subcutaneous zygomycosis.

Viral Panniculitis caused by cytomegalovirus[75] presenting as subcutaneous nodules on the lower limbs, mainly septal panniculitis with many cytomegalic inclusions within the endothelial cells.

Classically, in infective panniculitis there is a mixed pattern of septal and lobular involvement .Neutrophilic involvement of fibrous septa can occur, a feature that can rarely overlap with acute erythema nodosum.

In immunosuppressed patients, microorganisms are numerous and they may be identified in tissue sections with the routine haematoxylin eosin stain or with special stain. In patients with preserved immune response, the microorganisms are sparse and they cannot be identified with special stain. Diagnosis must be established by a positive culture of lesion or serological studies.

Subcutaneous zygomycosis[76, 77]

Zygomycosis broadly consist of Mucormycosis and Subcutaneous zygomycosis. Subcutaneous zygomycosis further classified into Conidiobolomycosis and Basidiobolomycosis.

Subcutaneous zygomycosis (Basidiobolomycosis) is caused by Basidiobolous Ranarum. Minor injuries and cuts are considered the probable routes by which fungus gain access to human tissue.

Basidiobolomycosis occurs most commonly in children and usual sites of involvement are buttock, thigh and limbs. Clinically the disease is characterized by well circumscribed firm to hard subcutaneous nodules which spread circumferentially around an extremity as well as both proximally and distally. Progressive over weeks and months is typical. Infection spreads along contiguous subcutaneous tissue rather than by haematogenous route and the lymph nodes are not involved .The mass is usually movable over the deep muscle. The edges are smooth, rounded, clearly defined and the mass can be raised up by inserting the finger underneath it and this is characteristic of basidiobolomycosis. The lesions are generally painless. Overlying skin is shiny, oedematous, scaly, hyperpigmented or normal. Ulceration is very rare. The tissue growth may be negative for fungal growth. This could be probably due to the fact that zygomycetous fungi have primitive coenocytic hyphae that will often be damaged and become non-viable during the biopsy procedure or by the chopping up or tissue grinding process in the laboratory.

Histopathologically, most characteristic finding is the presence of short aseptate hyphae surrounded by eosinophilic material within the granulamatous tissue, demonstrated by methenamine silver or periodic acid - Schiff stains. The perihyphal deposition of eosinophilic material, the ‘Splendore-Hoeppli’ phenomenon may be demonstrated. In early stages

the granuloma is composed of eosinophils and neutrophils. In older lesion histiocytes, epitheloid cells and lymphocytes predominate, multinucleate giant cells also appear. Ultimately granuloma is replaced by fibrosis.

Factitial Panniculitis[78-80]

Panniculitis produced by external agents usually as a consequence of injection of drugs or chemical substance. An important cause of factitial panniculitis is the subcutaneous injection of oily materials including mineral oil like paraffin or vegetable oils like cotton seed and sesame oils[78]. Panniculitis has been reported following the injection of numerous therapeutic agents like pethidine, pentazocine[79], povidine or cytokines like Interferon-Beta, Granulocyte colony – stimulating factor.

There are reports of panniculitis at the site of tetanus antitoxoid vaccination and antihepatitis vaccines[80].

Extravasation of cytostatic agents like antracyclines, vinca alkaloids, and taxanes cause severe panniculitis during antineoplastic chemotherapy.

The histological features are not always specific and depend on the cause. It is a mixed septal and lobular panniculitis which is associated with a prominent degree of acute inflammatory or granulomatous infiltrate till fibrosis ensues. Polarization of slide can identify the refractile foreign material causing panniculitis.

Sclerosing Panniculitis (Lipodermatosclerosis) [81-83]

Sclerosing panniculitis, also named lipodermatosclerosis, hypodermatitis sclerodermaformis, stasis panniculitis, or chronic indurated cellulites, is a relatively common form of long term chronic panniculitis, included in the mostly lobular panniculitis without vasculitis. It usually develops in middle aged or elderly patients more frequently in women with high body mass index, chronic venous insufficiency, and previous episode of thrombophlebitis and less often with arterial ischemia[81].

It begins as circumscribed painful, warm, erythematous and oedematous plaques involving one or both lower legs above the medial malleolus with minimal induration[82]. Later the affected areas develop a marked induration of wood like consistency and hyper pigmentation with a stocking distribution. Finally, the extensive deep fibrosis leads to atrophy of the subcutaneous fat, which results in a deformity of the leg that resembles an inverted bottle. The overlying skin may show changes of venous stasis in addition to hyperpigmentation and atrophy such as ulceration and telangiectasias.

Histologically, three stages of disease were noted by Jorizzo[83], namely early, intermediate, and advanced. The earliest lesions showed an infiltrate of lymphocytes in septa of subcutaneous fat, ischemic necrosis with ghosts of adipocytes devoid of nuclei, and congestion of centrilobular

capillaries with hemorrhage and deposits of hemosiderin. Intermediate lesions showed septa thickened by fibroplasia and a mixed infiltrate in which there were macrophages and plasma cells. Zones of "hyalinized sclerosis" extended from septa into the dermis, where stasis changes were associated with an infiltrate of lymphocytes. Advanced lesions were characterized by sclerosis of septa; infarcts of lobules that were replaced by fat "microcysts," and membranous fat necrosis.

In any stage of the process, changes of stasis dermatitis may be observed in papillary dermis like proliferation of capillaries, fibrosis, extravasated erythrocytes and hemosiderin deposition.

Panniculitis with vasculitis:

Leukocytoclastic Vasculitis[1]

It is a most common type of vasculitis mainly affecting the cutaneous post capillary venules, characterized by palpable purpura, urticaria or ulcers on the leg involving only small vessels. It some instances, lesion of cutaneous leukocytoclastic vasculitis appear in the form of subcutaneous nodules.

Histopathological study of these lesions demonstrates thickened septa with features of leukocytoclastic vasculitis involving small blood vessels. There is no involvement of the dermal blood vessels; usually there is little or no inflammatory infiltrate at the adjacent fat lobule.

Large Vessel Vasculitis:

Superficial thrombophlebitis [84, 85]

It presents with erythematous tender subcutaneous nodules arranged in a linear fashion with a cord like thickening of subcutis along the involved veins and is usually located on the lower limbs. Venous insufficiency of lower extremities is a known precipitating factor.

Histopathologically the cutaneous lesions of superficial migratory thrombophlebitis involve large veins of septa in upper subcutis. There is little or no involvement of adjacent fat lobule and the process is more vasculitic than panniculitis.

Cutaneous polyarteritis nodosa [86, 87]

An uncommon form of vasculitis runs a chronic but benign course. It involves small and medium sized arteries of dermis and subcutaneous tissue. It is also called periarteritis nodosa. The cause of cutaneous PAN is not known it could be a hypersensitivity reaction to certain infection[86]. Women were affected slightly more than men.

Cutaneous PAN is characterized by tender, subcutaneous nodules, usually measuring 4-5 mm in diameter, along with infarcts presenting as purple or black patches and livedoreticularis that may ulcerate[87].They are mostly formed over legs and feet. Histopathological examination shows an inflammatory necrotizing obliterative arteritis affecting small and medium

sized arteries with focal panniculitis. The most common histological findings was periarteritis of small to medium sized arterioles at dermal-pannicular junction.

Erythema induratum of Bazin: (Syn: Nodular Vasculitis) [88-91]

It is a chronic nodular eruption that usually occurs on the lower legs of young women. It was described by Bazin in 1855. Erythema induratum of Bazin has been regarded as a manifestation of tuberculosis hypersensitivity (ie a type of tuberculid occurring on the legs) [2]. In 1945 montgomery and colleagues[90] introduced the term "nodular vasculitis" to designate the lesions of erythema induratum of nontuberculous origin. In recent years, however, numerous works have demonstrated the presence of mycobacterium tuberculosis (MTB) DNA recovery by polymerase chain reaction from skin lesions of erythema induratum Bazin. Most authors currently consider Erythema induratum Bazin or nodular vasculitis as a multifactorial reactive disorder with many different causes with tuberculosis being one of them.

Clinically, the patients are usually young or middle age woman. They present with recurrent flares of violaceous nodules or deep seated plaques on the legs. The lesions are cold, not painful, and have a tendency to central ulceration. The superficial skin tends to show desquamation that forms a scaly collarets around the lesions or crusts overlying the ulcers.

Most lesions resolve spontaneously within few months, leaving postinflammatory hyperpigmentation and occasionally atrophic pigmented scars. The most frequent locations of these lesions are the post aspect (calves) and anterolateral areas of the legs, the feet, thigh, arms and face.

Histopathologically, erythema induratum of Bazin is a lobular panniculitis that shows a granulamatous inflammation with focal necrosis, neutrophilic vasculitis and septal fibrosis in varying combinations[88,89]. The granulamatous inflammatory infiltrate shows epitheloid cell, foamy histiocytes and giant cell that may be of the Langhan's type or foreign body type. In some cases extensive necrosis of panniculus with predominantly neutrophilic infiltrate forming true abscess can be seen. The presence of vasculitis in the specimen is not always identified and is not considered a requisite for making diagnosis[91]. In same cases with extensive panniculitis, the granulamatous infiltrate can extend to the deep reticular dermis. Special stains do not demonstrate the presence of acid fast bacilli. When intense vascular damage is present, extensive areas of caseous necrosis appear. Caseous necrosis may extend to the overlying dermis and secondary involvement of epidermis with ulceration and discharge of liquefied necrotic fat is reported.

Neutrophilic panniculitis[92]

It can occur in the early stages of many of the panniculitis. Causes of neutrophilic panniculitis include Alpha-1 antitrypsin deficiency panniculitis, pancreatic panniculitis, factitious panniculitis, neutrophilic panniculitis / subcutaneous sweet syndrome, neutrophilic / Pustular panniculitis of rheumatoid arthritis, erythema nodosum like lesions of Behcet's disease, Bowel by pass dermatosis, iatrogenic panniculitis.

Histopathologically, the infiltrate composed predominantly of mature polymorphonuclear leucocytes mostly involving the fat lobules, although secondarily the infiltrate may also extend into the adjacent connective tissue septa[92].

Oedematous scarring vasculitic panniculitis[93]

This can be identified in children. It represents an evolutionary process in the development of malignant lymphoma in children.

Erythema nodosum leprosum [94-99]

Erythema nodosum leprosum (ENL) or type II reaction is an immune complex syndrome seen in multibacillary leprosy[95]. ENL usually occurs after specific treatment of lepromatous disease or borderline leprosy but may be observed in patients who have not been treated.

ENL presents with inflammatory skin nodules and involvement of multiple organs, often running a protracted course. Over one half of

lepromatous leprosy patients and one quarter of borderline lepromatous patients will experience type 2 reaction[98].

The clinical manifestation is of evanescent crops of tender erythematous papules or nodules located on interlesional skin. They are all dome shaped with ill defined margins. The papules may turn into pustules or may simply ulcerate. They can also occur as subcutaneous plaques. The lesions are most common on the face and extensor surface of the limbs and less commonly on the trunk, accompanied by fever, malaise, arthralgia and leukocytosis. Lesions tend to recur at the same sites and if they do not resolve completely, a chronic painful panniculitis develops which may persist for months or years. Large areas of inflamed skin and subcutaneous tissue thus becomes fixed to underlying fascia, muscle and bone and may thus immobilize a hand or foot or even face[99].

Histopathology: In erythema nodosum leprosum, the lesions are foci of acute inflammation superimposed on chronic multibacillary leprosy[97]. It is characterized by oedema of papillary dermis and a mixed dermal infiltrate of neutrophils and lymphocytes superimposed on collection of macrophages. Vasculitis may also be present. There may be involvement of subcutis, with the development of a mixed lobular and septal panniculitis: however, in majority of case involvement of the dermis is the primary and predominant finding. Macrophages in dermis contain

fragmented organism. Polymorph neutrophils may be scanty or so abundant as to form a dermal abscess with ulceration .Whereas foamy macrophages containing fragmented bacilli are usual, in some patients no bacilli remain and macrophages have a granular pink hue on Wade-Fite staining , indicating mycobacterial debris. A necrotizing vasculitis affecting arterioles, venules and capillaries occurs in some cases of ENL; these patients may have superficial ulceration[97.]

Lucio's Phenomenon[100, 101]

Lucio's phenomenon is a vasculitis clinically described in 1852 and microscopically documented in 1948 in patients with diffuse lepromatous leprosy. Usually occurs in patients who have received either no treatment or inadequate treatment. In contrast to ENL, fever, tenderness and leukocytosis are absent. The lesions consist of barely palpable, haemorrhagic, sharply marginated, irregular plaques. They develop into crusted lesions and, particularly on the legs, into ulcers.

Histopathologically, Lucio phenomenon is a distinctive type of granulomatous and necrotizing panvasculitis[100] of small vessels in the upper and mid dermis that results in ulceration of epidermis. Occasionally the process extends deep into the subcutaneous fat with small vessel vasculitis in the fat lobule[101].

AIM OF THE STUDY

1) To study the clinical and histopathological features of various types of panniculitis.
2) To find out the common types of panniculitis by clinical and histopathological parameters.

MATERIALS AND METHODS

A study was conducted during the period from April 2008-October 2009 in the Department of Dermatology, Govt Rajaji Hospital, Madurai Medical College, Madurai, among the patients attending the dermatology department as well as those referred from other departments mainly Medicine, Surgery and Pediatrics.

Inclusion Criteria:

1. All patients with clinical features suggestive of panniculitis i.e erythematous subcutaneous nodules or plaques with or without ulceration which was subsequently confirmed by histopathological examination

Exclusion Criteria:

1. Patients who were unwilling for the study
2. Patients who were diagnosed clinically as panniculitis but the histopathological features not confirming the diagnosis of panniculitis.
3. Patients with abnormal bleeding parameters.

A proforma was filled for all patients

History:

A detailed history was taken which includes, duration of skin lesions, recurrent nature of lesion, presence or absence of pain, ulceration

and systemic symptoms. History of sore throat in the recent past, present or past history of tuberculosis, history of drug intake ,use of oral contraceptives, history suggestive of malignancy and collagen vascular disorders and occupational history.

Clinical Examination:

Detailed general and systemic examinations including rheumatological examination were done. Detailed examination of skin lesion which includes morphology of skin lesions, distribution of lesions, and number of lesions, symmetry, tenderness, ulceration, induration and size were all recorded.

Investigations:

1. Routine laboratory investigations included are complete haemogram, renal function test, liver function test, serum amylase, ASO titre and skin smears for acid fast bacilli
2. Screening for HIV, Hepatitis, and Syphilis were also done for high risk patients with history of sexual exposure or occupational exposure to blood and blood products.
3. Mantoux test : Mantoux test with 5 TU (Tuberculin units) was performed as a routine investigation in all patients

4. Radiological investigations included are chest X ray and U/S for all patients. Doppler studies in patients with suspected lipodermatosclerosis.
5. Tests to rule out malignancy and collagen vascular disorders were done when indicated.
6. Incisional elliptical skin biopsies were done from the representative skin lesions with a caution not to include the resolving lesion and they were sent to pathologist. Special stains like AFB were done by the pathologist when required.

Analysis:

A descriptive analysis of the clinical characteristics, laboratory parameters and histopathological features of various panniculitis was done. The data was analyzed and compared with published literature.

OBSERVATION AND RESULTS

Fifty three patients with clinical features of panniculitis were seen during the study period from April 2008 to October 2009. Of these, 17 cases were excluded from the study because of the patients denial for the study (4), not willing for biopsy (3) and patients who showed no histopathological features of panniculitis (10).

Thirty six patients with clinical and histological features of panniculitis were included in the study. The common types were EN (12) and ENL (6). The less common types were panniculitis associated with connective tissue diseases (4), EI (3) lipodermatosclerosis (2), panniculitis of arthropod bite (2), subcutaneous zygomycosis (2) and pancreatic panniculitis (1). Four patients could not be classified.

Table- 1

The clinical spectrum of panniculitis

Disease	**No. of Patients**	**Percentage**
Erythema Nodosum	12	33
Erythema nodosum leprosum	6	17
Erythema induratum	3	8
Lipodermatosclerosis	2	6
Others	13	36

Table -2

Sex Distribution

	Male		Female	
Disease	**No.**	**%**	**No.**	**%**
Erythema Nodosum	5	42	7	58
Erythema nodosum leprosum	3	50	3	50
Erythema induratum	1	33	2	67
Lipodermatosclerosis	2	100	0	0
Others	10	77	3	23
Total	21	58	15	42

In our study, 21 patients were male while 15 patients were female. Male, Female ratio is 1.4: 1 (58% & 42 %)

Table -3

Age Distribution

Age in years	EN	ENL	EI	LDS	Others	Total
< 10					2	2
10 – 20	2					2
21 – 30	5	1	1		2	9
31 – 40	4	2	2	1	5	14
41 – 50	1	2		1	2	6
51 – 60						
> 60		1			2	3
Total	12	6	3	2	13	36

Majority of the patients were between the ages of 21-50 yrs

The mean age of presentation was 35.5 yrs.

Thirty six cases with histopathological features of panniculitis were classified into four categories

Septal panniculitis - 12 (33 %)

Lobular panniculitis - 5 (14%)

Mixed panniculitis - 10 (28 %)

Panniculitis with vasculitis- 9 (25%)

Table -4 Septal panniculitis - Distribution of cases

Septal panniculitis	No. of cases	Percentage
EN	9	75
Eosinophilic fasciitis	1	8.3
Subcutaneous nodules of still's disease	1	8.3
Subcutaneous morphoea	1	8.3

Table – 5 Lobular panniculitis – Distribution of Cases

Lobular panniculitis	No.of cases	Percentage
Pancreatic panniculitis	1	20
Idiopathic	4	80

Table – 6 Mixed panniculitis – Distribution of cases

Mixed panniculitis	No. of cases	Percentage
Lipodermatosclerosis	2	20
Subcutaneous zygomycosis	2	20
Panniculitis of arthropod bite	2	20
LE Panniculitis	1	10
Erythema nodosum	3	30

Table – 7 Panniculitis with Vasculitis distribution of cases

Panniculitis with Vasculitis	No. of cases	Percentage
ENL	6	66.66
EI	3	33.33

DESCRIPTION OF CASES AND FINDINGS:

Erythema nodosum

There were 12 (33%) cases of erythema nodosum.

Clinical profile*:*

The age group of patients presented with features of erythema nodosum was between 14 to 45 years. The male to female ratio was 1: 1.4 The mean duration of the skin lesions was 5.5 weeks. Systemic symptoms of fever and arthralgia were present in seven (58%) of the patients. All the patients had tender nodules symmetrically distributed over the lower limbs (100%) (fig.1) Involvement of upper limbs and trunk was present in four (33%) patients in additions to the classical lesions over the lower limbs.

Abnormal investigatory findings:

The Mantoux test was positive in two (17%) patients. A high ASO titre was found (>300 Todd units/ml) in four (33%) of the patients. Widal test was positive in one patient.

Histopathology:

Among the twelve patients, nine (75%) patients showed features of septal panniculitis and three (25%) showed features of mixed panniculitis. The infiltrate was predominantly lymphohistocytic (10) and neutrophilic (2). Granulomas with in the septa were demonstrated in four (33%) of our cases.

Table -8

Erythema nodosum clinical features

Laboratory investigations and histopathology

Age	No. of Patients	DSL Weeks	Sys sym	Mx test (>10mm)	ASO (>300)	Widal Test	CXR (abn)	ANA	SP	MP
< 10										
10-20	2	3.5	1	1	1				1	1
21-30	5	9.4	3		3				3	2
31-40	4	7	2			1			4	
41-50	1	2	1	1					1	
51-60										
> 60										

Key - DSL = Duration of skin lesions, Sys sym = Systemic symptoms, Mx test = Mantoux test, CXR = Chest X ray, SP = septal panniculitis, MP = Mixed panniculitis

Erythema nodosum leprosum (ENL)

There were 16 patients presented with features of ENL. Among these 16 patients only 6 patients subsequently showed involvement of subcutaneous tissue histopathologically.

Clinical Profile

The age group of patients presented with features of erythema nodosum leprosum was between 24 years to 67 years. The male to female ratio was 1.6:1. The mean duration of illness was 15.5 weeks. Ten (63%) patients belong to the LL spectrum and six patients (37%) belong to the BL spectrum. Systemic symptoms of fever and joint pain were present in all cases. The lesions consisted of tender nodules and plaque which were predominantly distributed in the lower limbs of all patients. Additional involvement of the trunk in 10 (63%) patients and face in 9 (56%) patients. The lesions were symmetrically distributed in all cases. Five patients (31%) showed ulceration.

Abnormal Investigatory findings:

The BI ranging from 4 to 6 was present with fragmented and granular morphology. Mantoux test was positive in three patients (30%). Chest X ray showed evidence of tuberculosis in one patient (10%).

Table 9

Erythema Nodosum Leprosum -clinical features

Laboratory investigations and histopathology

Age	No.of Patients	DSL weeks	Sys sym	Mx test (>10mm)	ASO (>300)	CXR (abn)	ANA	P+V	LL	BL
< 10										
10-20										
21-30	3	13	3	2				2	3	
31-40	6	14.3	6	1				2	4	2
41-50	4	11	4					1	2	2
51-60	2	22	2					1	1	1
> 60	1	17	1			1				1

Key - DSL = Duration of skin lesions, Sys sym = Systemic symptoms, Mx test = Mantoux test, CXR = Chest X ray, SP = septal panniculitis, MP = Mixed panniculitis. P+V – Panniculitis with vasculitis, LL – Lepromatous leprosy, BL – Borderline lepromatous leprosy

Histopathology **:**

Among the sixteen patients with clinical features of ENL, six (37%) patients showed features of panniculitis with vasculitis. Mixed type of panniculitis in four (67%) patients and mostly lobular pattern of

panniculitis in two (33%) patients, characterized by infiltration of neutrophils, lymphocytes, dermal and subcutaneous macrophage granuloma with edema of papillary dermis. Features of vasculitis were present in seven (44%) patients. The dermal nerves were infiltrated by polymorphs. Ten (63%) patients showed inflammation only in the dermis (Hence excluded from this study of panniculitis.) Acid fast stain showed fragmented and granular acid fast bacilli.

Erythema induratum:

There were three (8 %) patients presented with features of erythema induratum

C***linical Profile***

The age group of patients presented with features of Erythema induratum was between 28 to 40 yrs. The male to female ratio was 1:2. The mean duration of skin lesion was 10 weeks. Systemic symptoms of fever and joint pains were present in two patients. Lesion consisted of tender, erythematous nodules and plaques on the posterior aspects of lower legs. The lesions were symmetrical in all patients. Two of the three patients showed ulceration. The lesions healed with scarring.

Abnormal investigatory findings:

Mantoux test was positive is two (67%) patients. Elevated ESR in all three patients

Histopathology:

All three patients showed evidence of lobular panniculitis with vasculitis and inflammatory infiltration of lymphocytes and neutrophils. Granulamatous inflammation composed of epitheloid histiocytes, lymphocytes and langhan's giant cells was noted in two (75%) patients.

Table 10

Erythema induratum clinical features

Laboratory investigations and Histopathology

Age	No.of Patients	DSL weeks	Sys sym	Mx test (>10mm)	ASO (>300)	CXR (abn)	ANA	LP	MP	P+V
< 10										
10-20										
21-30	1	18	1	1				1		1
31-40	2	22	2	1				2		2
41-50										
51-60										
> 60										

Key - DSL = Duration of skin lesions, Sys sym = Systemic symptoms, Mx test = Mantoux test, CXR = Chest X ray, SP = septal panniculitis, MP = Mixed panniculitis. P+V – Panniculitis with vasculitis.

Lipodermatosclerosis

There were two (6%) cases of lipodermatosclerosis (Sclerosing panniculitis)

Clinical profile

The mean age of presentation was 43 years. Both the patients were male. The duration of skin lesions ranged between 6-12 months. Cutaneous lesions consist of diffuse, painful, warm, indurated plaques involving lower legs symmetrically above the medial malleolus in both patients. The overlying skin showed changes of venous insufficiency in addition to the hyper pigmentation and clinical evidence of varicosity in both patients.

Abnormal investigatory findings:

Doppler ultra sonogram study showed features of venous insufficiency in both patients

Histopathology

Skin biopsy showed mixed panniculitis with septal thickening and a mixed cell infiltrate in which there were macrophages and plasma cells.

Panniculitis of Arthropod stings

There were two cases under this category

***Clinical Profile*:** A 55 year old male presented with tender erythematous plaque lesion of size 12x10 cm over the right chest just above the right nipple. Few vesicles and bullae present overlying the plaque.

Another patient, a 61 year old male admitted with tender erythematous, oedematous plaque lesion of size 10x8 cm extending from the lower arm to upper forearm with overlying vesicles and bullae.

Both these patients had a history of insect bite followed by occurrence of skin lesion over the night and residual indurated plaque lesion after the treatment of acute phase.

***Abnormal investigatory findings*:**

Both the patients had elevated ESR, and total leukocyte count. Second patient had elevated renal parameters.

Histopathology:

The skin biopsies were done after the acute phase from the plaque lesion. There was a chronic lymphoid response with dense lymphoplasmocytic inflammatory infiltrate in the dermis extended into the subcutaneous plane. Subcutis showed mixed panniculitis with inflammatory infiltrate of lymphohistiocytes with an admixture of eosinophils and plasma cells.

Pancreatic Panniculitis:

***Clinical Profile*:**

A 45 year old male patient, a chronic alcoholic presented with erythematous tender subcutaneous nodules distributed symmetrically over the lower legs of 4 weeks duration. Few lesions healed with depressed

scars. The patient was admitted in Medicine Department for the complaints of fever, abdominal pain and joint pain .Subsequently he was labeled as a case of pancreatitis.

***Abnormal investigatory findings*:**

Patient had intermittent elevation of serum amylase (140-200 u/l), leukocytosis (14,500 leukocytes / cu mm) and elevated hematocrit. Ultra sound abdomen showed features of pancreatitis.

***Histopathology*:**

Skin biopsy showed mostly lobular panniculitis with predominant neutrophilic infiltrate and necrosis of adipocytes. Deposition of homogenous basophilic material within the fat lobules, suggestive of calcium salts.

Eosinophilic fasciitis:

A 28 yr old male presented with diffuse indurated plaque lesion involving both forearms symmetrically of six months duration, associated with mild pain and tenderness. He had no systemic symptoms.

Abnormal investigatory findings:

The patient had elevated ESR and blood eosinophilia.

Histopathology

Skin biopsy showed features of septal panniculitis without vasculitis, thickening of deep fascia and septa of subcutis with fibrosis and

hyalinization of collagen with inflammatory infiltration of septa and deep fascia with lymphocytes, neutrophils, plasma cells and eosinophils. Few inflammatory infiltrate also extending into deep dermis.

Lupus erythematosus panniculitis:

A forty year old female patient presented with tender indurated subcutaneous nodules and plaques involving the right arm, both thigh and buttock regions of one month duration, associated with joint pain ,fever and oral lesions. History of similar lesions four years back. This patient developed extensive ulceration of the skin over the lesions on the buttock and thigh region in the subsequent month.

Abnormal investigatory findings:

This patient had elevated ESR, anemia, positive anti-double-stranded DNA antibody titre.

Histopathology

Skin biopsy showed a picture of mixed panniculitis with predominant lobular involvement. There was a lobular and periseptal lymphocytic infiltration with necrosis of fat lobules.

Subcutaneous Morphoea

A 30 year old female patient presented with solitary indurated plaque lesion over the back of trunk of 6 month duration. Patient is asymptomatic.

***Abnormal investigatory findings*:**

Investigations in this patient did not reveal any abnormality.

***Histopathology*:**

Skin biopsy showed septal panniculitis and dermal sclerosis with lymphoplasmocytic infiltration.

Subcutaneous nodules of Juvenile Rheumatoid arthritis:

Clinical Profile:

An 8 year old girl presented with erythematous tender subcutaneous nodules discretely present over the left forearm, right arm, left shoulder and in right leg of 3 weeks duration. She had joint pain involving small joints of 6 month duration.

Abnormal investigatory findings:

Investigations revealed a positive rheumatoid factor and increased ESR.

The histopathology showed fibrosis of dermis with minimal inflammatory infiltration of subcutaneous tissue. Patient was started on oral steroids with which there was a good response and the skin lesions were resolved.

Panniculitis of deep fungal infection:

There were two cases of Subcutaneous Zygomycosis

A three year old boy presented with firm, painless, gradually progressive skin lesion on his left thigh of six months duration. On examination,

patient had solitary, firm, non tender subcutaneous plaque lesion of size 8x5 cm with areas of scarring and depression in the centre.

Another patient, a 38 year old male presented with pigmented , indurated plaque lesion of size 10x8 cm over the lateral aspect of right elbow of eight month duration. History of topical application of native medicine followed by ulceration over the plaque lesion. The routine laboratory parameters of both these patient were normal.

Histopathology:

The skin biopsy of the first patient showed mixed panniculitis. Inflammatory infiltrate composed of predominant eosinophils and giant cells in H&E stain. The tissue growth was negative for fungal growth after 7 days.

Biopsy of the second patient showed dense subcutaneous inflammation with predominant eosinophilic infiltration.

Both these patients responded well with antifungal drugs . Diagnosis based on the histopathological features and response to oral antifungal drugs but not by any other antibiotics.

HIV associated lipodystrophy:

There were two cases of HIV associated lipodystrophy.

First case, a 45 year old male, diagnosed as a case of AIDS, started on antiretroviral therapy with stavudine, lamivudine and nevirapine regimen

eight months back. The patient presented with localized lipodystrophy of face with loss of buccal, parotid and preauricular pad of fat, resulting in prominent zygomata, sunken eyes, and cachectic facies.

Another patient, a 60 year old female, diagnosed as a case of AIDS five years back on treatment with antiretroviral drugs (Stavudine, Lamivudine, nevirapine) of one year duration, presented with increased fat deposition in the dorsocervical regions (buffalo hump) and localized lipodystrophy involving the face.

Both patients are HIV reactive with CD4 count of less than 200 cells /cu mm.

These two patients were not willing for biopsy, so the diagnosis was based on history, clinical features and laboratory parameters only

Four patients with lobular panniculitis remained unclassified as the clinical features, etiology and histopathology were not suggestive of any specific form of panniculitis.

DISCUSSION

The panniculitides are a diverse group of cutaneous disorders that are characterized by an inflammatory process that predominantly affects the subcutaneous fat. Clinically most of the patients with suspected panniculitis were presented with nonspecific features of erythematous, tender, subcutaneous nodules usually located in the lower limbs. Histopathologically there may be overlap between the various forms of panniculitides. Deep incisional or excisional biopsy and serial biopsies were required for the conclusive diagnosis.

One of the largest published clinicopathological study on panniculitis was by Handa et al[10] between 1997-2001 in All India institute of medical science, New Delhi. They included 29 patients .The results of this study by Handa et al[10] indicated that the mean age of presentation of patients with panniculitis was 33.5 years, females comprised nearly 83 % of cases. Panniculitis were rare in children. Septal panniculitis without vasculitis was the most common histopathologic pattern observed. Erythema nodosum was the commonest type of panniculitis accounting for 23 cases (79.3 %).Elevated ESR and leukocytosis were the commonly observed laboratory abnormalities in that study .

In our study, the mean age of presentation of our patients was 35.5 years (3- 61years) with a rare incidence in children. Males were more commonly affected in our study, whereas in the study by Handa et al[10]

females were more commonly affected. The most common type of panniculitis in our study was erythema nodosum consistent with the study by Handa et al[10]. Other types of panniculitides that we encountered were erythema nodosum leprosum, lipodermatosclerosis and erythema induratum of Bazin. The rare types of panniculitides in our study were pancreatic panniculitis, subcutaneous zygomycosis eosinophilic fasciitis and panniculitis of arthropod bite.

Clinical profile of erythema nodosum was similar to other studies[10,11]. In our study, mean duration of skin lesions was 5.5 weeks, where as in other studies the duration varied from 3 weeks to 33.8 weeks. Females were more commonly affected, consistent with other studies[10, 11]. In the study by Handa et al, erythema nodosum was caused by Behcet's syndrome in 3 (13.1%) cases, sarcoidosis in 4 (17.4%) cases and idiopathic in 16(69.5%) of cases. In the study from west by Cribier et al[11], infections were the most common cause of erythema nodosum with streptococcal infection occurred in 28%, chlamydial infection in 1.5 %, mycoplasmal infection in 0.8% . In our study 58% of patients had evidence of infection which included streptococcal infection in 33%, tuberculosis in 17% and salmonella infection in 8%, the rest of the case were idiopathic (42%). The drugs were not implicated as a cause of erythema nodosum. There was no seasonal variation seen in our study unlike that reported by Handa et al. Histopathologically septal panniculitis

without vasculitis was the most common type , comprising 75% of cases and the remaining 25% of cases were mixed panniculitis where as in the study by Handa et al[10], all cases of erythema nodosum showed septal panniculitis. Macrophage granulomas were seen in the septa in four of our cases. Late lesions of erythema nodosum may show widened septa, often with fibrosis and inflammation at the edges of the septa and involving the periphery of fat lobules that constitute a mixed panniculitis. Macrophage granulomas without lipid deposition are more frequent when late lesions are compared to early ones. The acute onset of lesions over the lower limbs and associated arthralgia in most of our patients were consistent with the study done by Handa et al[10].

ENL was described as Panniculitis nodosa leprosa by W.J.Peplar [94] in 1955. Clinically individual lesions resemble erythema nodosum. However, there are few differences in the overall clinical picture. ENL affects areas such as face, which is seldom affected in EN and it covers larger areas the body. The lesions over the face were seen in 56% of the patients in our study. Disseminated lesions over the trunk and upper limbs were present in 63% of patients and 31% had ulcerated lesions. In the study of histopathology of ENL by Peplar et al[94], 18(78.2%) out of 23 specimens showed a mixed panniculitis and 3(13%) specimens showed lobular panniculitis. In our study four (25%) patients showed mixed type of panniculitis with vasculitis

and two (12.5%) patients showed mostly lobular pattern of panniculitis with vasculitis. In our study 63% of patients clinically diagnosed as ENL showed only dermal pathology. Although ENL patients presented clinically suggesting subcutaneous nodules and plaques only 37 % of patients were subsequently found have involvement of subcutaneous tissue histopathologically in our study which is consistent with the fact that the involvement of the dermis is the primary and predominant finding in majority of cases, as reported in standard literature[96]. Features of vasculitis were present in 44% of our patients. ENL with vasculitis is more common in India as reported by Giam YC et al[95].

Erythema induratum of Bazin was initially described by Bazin in 1861 and considered as a distinct entity caused by tuberculous infection[88].

The prevalence of erythema induratum in our study was higher (8%) than that reported by Handa et al(3.12 %) .The clinical features were similar to that reported from a study done from south east Asia[88]. Erythema induratum is considered to have multiple etiologies, tuberculosis being one of them[89]. In our study 66% of patients had positive Mantoux test. The predominant histopathological pattern observed was lobular panniculitis as reported earlier. Most of the patient in our study showed features of vasculitis consistent with the study by Segura et al[91].

The study from West showed that lipodermatosclerosis is more prevalent in elderly women[81], but in our study this entity was seen in men alone. Most of the clinical features were consistent with the study done earlier except erythema. The prevalence of erythema reported was 71% in the study by Bruce et al[81], whereas it was not seen in our study. Erythema may not be appreciable in darkly pigmented skin of our Indian population.

Panniculitis of arthropod bite is a rare event described in two of our patients. Eosinophilic panniculitis due to arthropod bite was reported by Adame J et al[16] and it is considered as a reactive process due to various causes, one of them being arthropod bite. Our patients had history of arthropod bite followed by appearance of skin lesion in the form of vesicles and blisters that resolved with painful residual plaque lesion. Histopathology showed mixed panniculitis with inflammatory infiltrate of lymphohistiocytes with an admixture of eosinophils and plasma cells. It is a chronic lymphoid response in the dermis extending into the subcutaneous plane. The patchy infiltrate of lymphocytes and eosinophils in the subcutaneous fat, like that in the dermis, is typical of a response to an assault by an arthropod.

Eosinophilic fasciitis, the prototype of the fasciitis-panniculitis syndrome (FPS) was rarely reported in Indian studies. In the study from west by Naschitz et al[20], most of the cases of FPS were idiopathic. Our patient had lesion distributed symmetrically over both forearms with histopathological

features of septal panniculitis without vasculitis. Thickening of deep fascia and interlobular fibrous septa of subcutis with infiltration of lymphohistiocytes, focal collections of eosinophils with fibrosis and hyalinization of collagen extending from dermis to subcutis were noted. These features were consistent with the study by Naschitz et al[20]. It should be differentiated from morphoea by its sudden onset, usual limitation to the structures underlying the skin and its tendency to resolve spontaneously[21].

We had one patient of subcutaneous Morphoea with histopathological features of septal panniculitis without vasculitis. The subcutaneous morphoea can be generalized or localized. The localized variant was seen in our patient. The term subcutaneous morphoea and morphoea profunda are often used interchangeably, although morphoea profunda has been proposed as the appropriate diagnosis when both subcutaneous fat and fascia are involved and subcutaneous morphoea for cases with involvement of fat only[17]. The septal fibrosis and hyalinization are the obvious features.

Lupus erythematosus panniculitis is an unusual clinical variant of lupus erythematosus which involves the deep dermis and subcutaneous fat. Described first by Koposi[57] in 1883, his finding was subsequently recognized as LE profundus, more accurately LE panniculitis. Lupus panniculitis mostly occurs in adult women[59]. In the study by Marten PB et al[59], showed that the lupus panniculitis is a chronic inflammatory disease of subcutaneous tissue

that can develop during the course of SLE, although most patients do not develop systemic lupus[59]. Our patient is a 40 year old female having recurrent flare-ups of skin lesions in the form of plaques which forms necrosis and ulceration in certain areas with periods of long remission. Histopathology showed a mixed panniculitis with lymphocytic infiltration and hyaline fat necrosis, consistent with the study by Ng PP et al[55]. Positive anti Double stranded DNA antibodies in this patient was an unusual finding. We could not do immunofluorescence as we lacked this facility.

There are only few case reports and studies regarding subcutaneous zygomycosis in India. In the largest study conducted by Thappa et al[77], total of 12 cases of Basidiobolomycosis were recorded and the diagnosis was established by direct KOH (10%) preparation of the biopsy specimen and by typical histopathological features (subcutaneous inflammation with predominant eosinophils, giant cells and demonstration of fungal elements) [77]. Main conclusions drawn from Indian data are that Basidiobolomycosis is predominantly a disease occurring in south India[77], usually occurs in children and usual sites of involvement are buttocks, thighs and limbs[76]. Our patients presented with long history of painless swelling that forms indurated plaque lesions over the lower limb and upper limb respectively, not responding with any broad spectrum antibacterial but responded very well with oral antifungal drugs. The tissue growth was negative for fungal growth after 7 days. This

could be probably due to the fact that zygomycetous fungi have primitive coenocytic hyphae that will often be damaged and become non-viable during the biopsy procedure or by the chopping up or tissue grinding process in the laboratory. Definitive diagnosis usually depends upon the histological examination of biopsy specimen[77]. Minor injuries and insect bites are considered the probable routes by which the fungus gains access to human tissues[76].

In the study by Bywaters EGL et al[22], Subcutaneous nodules of Juvenile rheumatoid arthritis closely resembled those from patients with rheumatic fever and, apart from a slightly greater frequency of fibrosis, did not resemble those from adults with rheumatoid arthritis. This study showed occurrence of rashes six times more frequently in still's disease. The Rose - Waller test was more preponderantly positive in patients of stills disease with nodules than in patients without nodules. Our case was a eight year old girl, a known case of juvenile rheumatoid arthritis presented with subcutaneous nodules, positive rheumatoid factor and elevated ESR. This patient responded well with anti-inflammatory drugs.

Lobular panniculitis at times may be a sign of underlying systemic disease. The syndrome of pancreatitis and lobular panniculitis occurs predominantly in males with alcohol associated pancreatitis and pancreatic carcinoma, due to the release of pancreatic enzymes, occurring in 2-3% of all

patients with pancreatic disorder[36]. In the study conducted by Dahl et al[37] subcutaneous fat necrosis preceded the diagnosis of pancreatic disease by 13 weeks .Our case was a 45 year old male, a chronic alcoholic presented with skin lesions over the pretibial area prior to the onset of signs and symptoms of pancreatitis with histopathological features of lobular panniculitis, consistent with the findings described by Johnson et al[36]. The cutaneous lesions of pancreatitis induced panniculitis are indistinguishable from idiopathic lobular panniculitis[39]. In 40% of cases associated with pancreatitis induced subcutaneous fat necrosis, the skin lesions are the presenting features[39]. A deep skin biopsy and demonstration of lobular panniculitis with characteristic 'ghost cells' is the most useful diagnostic test[38]. Awareness, that panniculitis may be the presenting feature of pancreatic disease is very important to timely diagnosis these patients and possible necessary intervention.

A lipodystrophy can be seen in patients with acquired immunodeficiency syndrome. Lipodystrophy of our patients occurred following the use reverse transcriptase inhibitors. It usually occurs following the use of protease inhibitors characterized by peripheral lipodystrophy and central adiposity[53].

SUMMARY

A prospective study of the clinical and histopathological features of panniculitis was done for a period of 19 months (April 2008-october 2009). Thirty six patients with clinical and histopathological features of panniculitis were included in the study. The average age at presentation was 35.5 years. The male, female ratio was 1.4:1. The common type of panniculitis included Erythema nodosum (12) and Erythema nodosum leprosum (6). The less common types being panniculitis associated with connective tissue diseases(4), Erythema induratum(3) lipodermatosclerosis (2), panniculitis of arthropod bite (2) ,subcutaneous zygomycosis(2) and pancreatic panniculitis(1). Clinically most of the patients with suspected panniculitis were presented with non specific features of erythematous tender subcutaneous nodules, usually located in the lower limbs. Septal panniculitis without vasculitis was the most common histopathological pattern observed. The clinical and histopathological profile of erythema nodosum was similar to other studies. Most patients of erythema nodosum had evidence of infection in our study. All the cases of lipodermatosclerosis occurred in men in our study which is in contrast to other published studies from the west. We found a significant proportion of patients with ENL presenting as panniculitis in our study.

CONCLUSION

1. Clinicopathological correlation is necessary for the diagnosis and classification of panniculitides.
2. Commonest presentation of panniculitis in our study was subcutaneous nodules in 47% of patients followed by plaque lesions in 30 % and 17% of patients presented with nodules and plaques. Lesions with ulceration were less common (2%).
3. The mean age of presentation was 35.5 years with male female ratio of 1.4:1. All the cases of lipodermatosclerosis were seen only in men in our study.
4. Erythema nodosum and Erythema nodosum leprosum were the commonest type of panniculitis observed in our study
5. Histopathologically, septal panniculitis without vasculitis was present in 33% of cases, mixed panniculitis in 28%, panniculitis with vasculitis in 25% and lobular panniculitis in 14% of cases.
6. Significant proportion of patients with ENL presenting as panniculitis in our study is in consistence with other Indian studies.
7. Septal panniculitis without vasculitis was the common histopathological pattern of panniculitis observed in our study.
8. The unusual variants of panniculitis like panniculitis of arthropod bite, subcutaneous zygomycosis and pancreatic panniculitis were seen in our study.

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GLOSSARY
CODE FOR MASTER CHART

NAD	–	No abnormality detected
A	-	Abnormal
N	–	Negative
P	-	Positive
0	–	Absent
P	–	Present
1	-	Papule
2	-	Nodule
3	-	Plaque
4	-	Nodules and plaque
5	-	Ulceration
6	–	Plaque and ulceration
NR	–	Non reactive
R	–	Reactive
SP	–	Septal panniculitis
LP	–	Lobular panniculitis
MP	–	Mixed panniculitis
V	-	Vasculitis
EN	–	Erythema nodosum
ENL	–	Erythema nodosum leprosum
EI	–	Erythema induratum
LDS	–	Lipodermatosclerosis
SUM	–	Subcutaneous Morphoea
SJRA	–	Subcutaneous nodules of juvenile rheumatoid arthritis
EF	-	Eosinophilic Fasciitis
PP	–	Pancreatic panniculitis
ID	–	Idiopathic panniculitis

HLD	–	HIV Lipodystrophy
SUZ	–	Subcutaneous zygomycosis
PAB	-	Panniculitis of Arthropod bite
LEP	–	Lupus erythematosus panniculitis
TOS	–	Type of skin lesion
REC	–	Recurrence
TEN	–	Tenderness
ULC	–	Ulceration
SCAR	–	Scarring
SYM	-	Symmetry
LL	–	Lower limb
UL	–	Upper limb
FACE	-	Facial lesions
TRU	-	Trunk lesions
VARIC	-	Varicose veins
SY S	-	Systemic symptoms
CXR	-	X- Ray chest
WIDAL	-	Widal test for typhoid
RF	-	Rheumatoid factor
ANA	-	Anti nuclear antibodies
ADNA	-	Anti double stranded DNA antibodies
USD	-	Ultrasound /Doppler ultrasonography
HIV	-	Human Immunodeficiency Virus
SAM	-	Serum amylase
SM	-	AFB Smear for Acid Fast Bacilli
CD	-	Clinical Diagnosis
PD	-	Pathological Diagnosis
ASO	-	Antistreptolysin O

PROFORMA

Name : Age : Sex :
Hosp .No : S.I .No :
Presenting complaints :
Duration of skin lesions
(in weeks)
Type of skin lesions : Papule
Plaque
Nodule
Others
Tenderness : Yes /No
Ulceration : Yes / No
Number of lesions :
Symmetrical : Yes /No
Site : Single /Multiple
Description :
-Scarring : Yes/No
-Others
Description
Systemic symptoms : Yes/No
Fever : Yes /No
Duration :
Arthralgia : Yes/No
Duration :

General examination
Weight :
Pallor : Yes /No
Temperature :
-LNE : Yes/No
Description :
- Arthritis : Yes/No
Description
Varicose veins : Yes /No
Description
Systemic examination
CVS :
Resp :
CNS :
GIT :
Investigations

Complete haemogram
LFT
S. Bilirubin
SGOT
SGPT
Alk phosp
A/G Ratio
Sr.Creatinine
VDRL
HBsAg
Aso titre
HIV
HCV
Skin smear for AFB
ANA
MANTOUX TEST
U/S Doppler

Skin biopsy

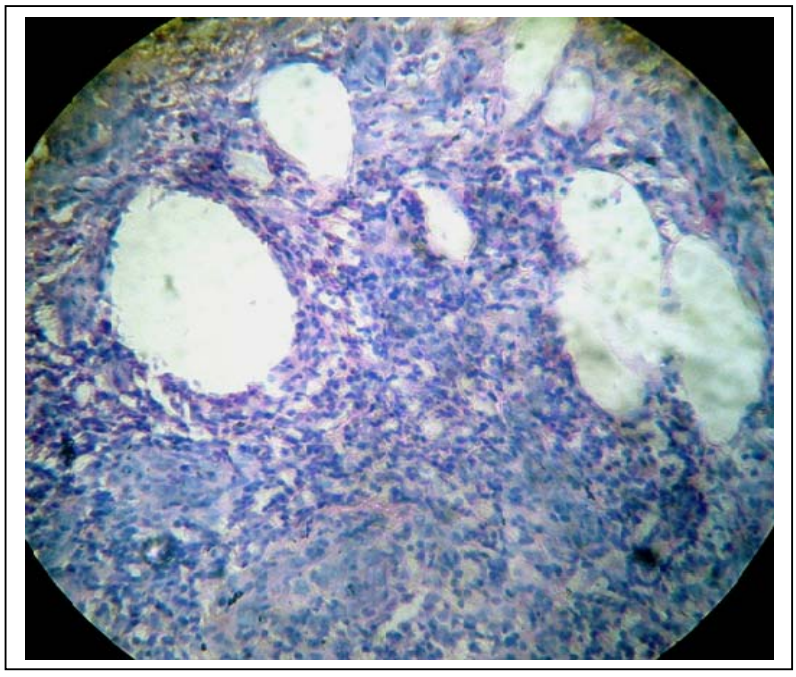
PANNICULITIS	**ABSENT**	**PRESENT**
Septal panniculitis		
lobular panniculitis		
Mixed panniculitis		
Granulomas		
Fat necrosis		
Vasculitis		
Small vessel		
Medium vessel		
Large vessel		
Epidermal changes		
Dermal changes		

Special stain
1.
2.
3.
Others
1.
2.
3.
Final Diagnosis : clinical Final Diagnosis: Pathological

S. No	Name	Age	Sex	TOS	REC	TEN	ULC	SCAR	SYM	LL	UL	FACE	TRU	VARIC	SY S	CXR	WIDAL	ASO	MX T	RF	ANA/ADN A	USD	HIV	S AM	SM AFB	CD	PD
1	Bose	35	M	4	1	1	0	0	1	1	1	1	0	0	1	NAD	N	N	N	N	N	NAD	NR	N	P	ENL	LP+V
2	Eswari	14	F	2	1	1	0	0	1	1	0	0	0	0	1	NAD	N	P	N	N	N	NAD	NR	N	N	EN	SP
3	Thangapandi	43	M	3	1	1	0	0	0	0	0	0	1	0	1	NAD	N	N	N	N	N	NAD	NR	N	N	IP	LP
4	Muthu	64	M	3	1	1	0	0	0	0	0	0	1	0	0	NAD	N	N	N	N	N	NAD	NR	N	N	IP	LP
5	Sumathy	42	F	2	1	1	0	0	1	1	1	1	0	0	1	NAD	N	N	N	N	N	NAD	NR	N	P	ENL	LP+V
6	Pitchipandi	40	M	2	1	1	0	0	1	1	0	0	0	0	0	NAD	N	N	N	N	N	NAD	NR	N	N	EN	SP
7	Vaitheswari	8	F	2	0	0	0	0	0	1	1	0	0	0	1	NAD	N	N	N	P	N	NAD	NR	N	N	SJRA	SP
8	Karupayee	40	F	2	1	1	1	1	1	1	0	0	0	0	1	NAD	N	N	P	N	N	NAD	NR	N	N	EI	LP+V
9	Seethalakshmi	25	F	2	1	1	0	0	1	1	0	0	1	0	1	NAD	N	N	N	N	N	NAD	NR	N	N	EN	SP
10	Sumathy	22	F	2	0	1	0	0	1	1	1	0	0	0	1	NAD	N	P	N	N	N	NAD	NR	N	N	EN	SP
11	Paramasivam	45	M	4	0	1	1	0	1	1	0	0	0	0	1	NAD	N	N	N	N	N	A	NR	P	N	PP	LP
12	Palanisanjeevaku	34	M	2	0	1	0	0	1	1	0	0	1	0	0	NAD	N	N	N	N	N	NAD	NR	N	N	EN	SP
13	Chandran	44	M	2	1	1	1	0	1	1	1	1	0	0	1	NAD	N	N	N	N	N	NAD	NR	N	P	ENL	D ENL
14	Parthiben	39	M	2	1	1	0	1	1	1	0	0	0	0	1	A	N	N	N	N	N	NAD	NR	N	N	EI	LP+V
15	Sivan	32	M	2	1	1	1	0	1	1	1	1	0	0	1	NAD	N	N	N	N	N	NAD	NR	N	P	ENL	D ENL
16	Ramu	52	M	3	1	1	0	0	0	0	0	0	1	0	1	NAD	N	N	N	N	N	NAD	NR	N	N	IP	LP
17	Vellaisamy	30	M	2	1	1	0	0	1	1	1	0	1	0	1	NAD	N	P	N	N	N	NAD	NR	N	N	EN	MP
18	Rajendran	40	M	2	1	1	1	0	1	1	1	1	0	0	1	NAD	N	N	N	N	N	NAD	NR	N	P	ENL	D ENL
19	Karthick	3	M	3	0	0	1	0	0	1	0	0	0	0	0	NAD	N	N	N	N	N	NAD	NR	N	N	SCZ	MP
20	Menaka	38	F	4	1	1	1	0	1	1	1	1	0	0	1	NAD	N	N	N	N	N	NAD	NR	N	P	ENL	D ENL
21	Mayandi	58	M	3	1	1	0	0	0	1	0	0	1	0	1	NAD	N	N	N	N	N	NAD	NR	N	N	IP	LP
22	Karuppu	55	M	3	0	1	0	0	0	0	0	0	1	0	1	NAD	N	N	N	N	N	NAD	NR	N	N	PAB	MP
23	Selvi	30	F	4	1	1	0	0	1	1	0	1	0	0	1	NAD	N	N	P	N	N	NAD	NR	N	P	ENL	MP+V
24	Muthu	24	M	2	1	1	1	0	1	1	1	1	0	0	1	NAD	N	N	N	N	N	NAD	NR	N	P	ENL	D ENL
25	Dhanalakshmi	30	F	3	0	0	0	0	0	0	0	0	0	0	0	NAD	N	N	N	N	N	NAD	NR	N	N	SM	SP
26	Kalpana	45	F	2	1	1	1	0	1	1	1	1	0	0	1	NAD	N	N	N	N	N	NAD	NR	N	P	ENL	D ENL
27	Velusamy	61	M	3	3	1	0	0	0	0	1	0	0	0	1	NAD	N	N	N	N	N	NAD	NR	N	N	PAB	MP
28	Raja	16	M	2	0	1	0	0	1	1	1	0	0	0	0	NAD	N	N	P	N	N	NAD	NR	N	N	EN	MP

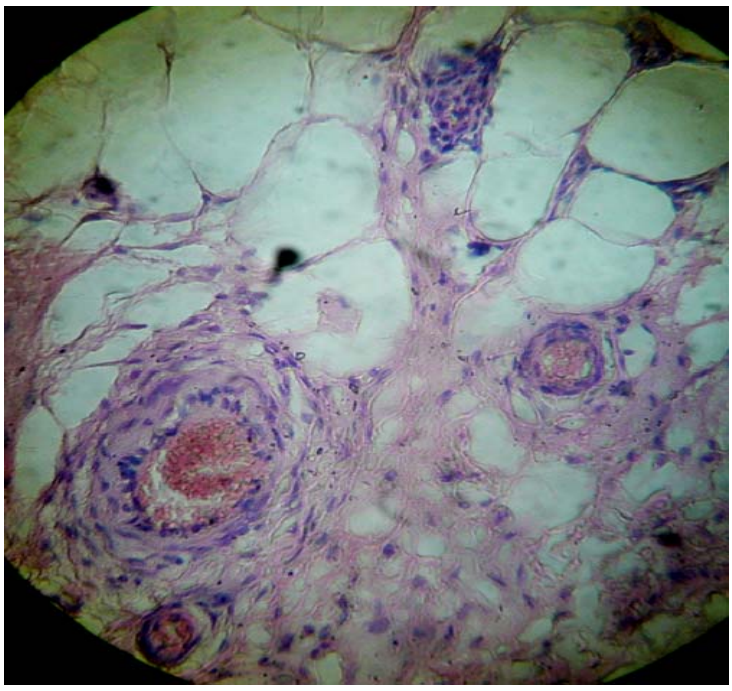
29	Muneswari	38	F	2	1	1	0	0	1	1	0	0	1	0	1	NAD	P	N	N	N	N	NAD	NR	N	N	EN	SP
30	Malar	32	F	2	1	1	0	0	1	1	0	0	1	0	1	NAD	N	N	P	N	N	NAD	NR	N	P	ENL	D ENL
31	Paulthurai	45	M	2	0	1	0	0	1	1	1	0	0	0	0	NAD	N	N	P	N	N	NAD	NR	N	N	EN	SP
32	Muthammal	25	F	2	1	1	0	0	1	1	0	0	0	0	1	NAD	N	P	N	N	N	NAD	NR	N	N	EN	SP
33	Panchavarnam	67	F	4	1	1	0	0	1	1	1	0	1	0	1	A	N	N	N	N	N	NAD	NR	N	P	ENL	MP+V
34	Valliammal	26	F	2	0	1	0	0	1	1	0	0	0	0	1	NAD	N	N	N	N	N	NAD	NR	N	N	EN	MP
35	Selvi	28	F	2	1	1	0	0	1	1	0	0	0	0	0	NAD	N	N	N	N	N	NAD	NR	N	N	EN	SP
36	Rajagopal	42	M	4	1	1	1	0	1	1	1	1	1	0	1	NAD	N	N	N	N	N	NAD	NR	N	P	ENL	LP+V
37	Krishnaswamy	49	M	3	0	1	0	0	1	1	0	0	0	1	0	NAD	N	N	N	N	N	A	NR	N	N	LDS	MP
38	Rajan	25	M	2	1	1	0	0	1	1	1	0	0	0	1	NAD	N	N	N	N	N	NAD	NR	N	P	ENL	D ENL
39	Lakshmannan	46	M	3	0	1	0	0	1	1	0	0	0	1	0	NAD	N	N	N	N	N	A	NR	N	N	LDS	MP
40	Natchiappan	38	M	2	1	1	1	0	1	1	1	1	1	0	1	NAD	N	N	N	N	N	NAD	NR	N	P	ENL	LP+V
41	Selvam	52	M	3	0	1	0	0	1	1	0	0	0	1	0	NAD	N	N	N	N	N	A	NR	N	N	LDS	-
42	Eswar	28	M	2	1	1	0	0	1	1	1	1	0	0	1	NAD	N	N	P	N	N	NAD	NR	N	P	ENL	D ENL
43	Perumal	60	M	4	1	1	1	0	1	1	1	1	0	0	1	NAD	N	N	N	N	N	NAD	NR	N	P	ENL	D ENL
44	Manikam	28	M	3	0	0	0	0	1	0	1	0	0	0	0	NAD	N	N	N	N	N	NAD	NR	N	N	EF	SP
45	Karupiah	45	M	3	0	0	0	0	0	0	0	1	0	0	1	NAD	N	N	N	N	N	NAD	R	N	N	HLD	-
46	Mariappan	38	M	3	0	0	1	0	0	0	1	0	0	0	0	NAD	N	N	N	N	N	NAD	NR	N	N	SCZ	MP
47	Krishnammal	28	F	4	1	1	1	0	1	1	0	0	0	0	0	NAD	N	N	P	N	N	NAD	NR	N	N	EI	LP+V
48	Indhirani	60	F	3	0	0	0	0	0	0	0	1	1	0	1	NAD	N	N	N	N	N	NAD	R	N	N	HLD	-
49	Revathy	40	F	6	1	1	1	1	0	1	1	0	1	0	1	NAD	N	N	N	N		NAD	NR	N	N	LEP	MP

ERYTHEMA NODOSUM



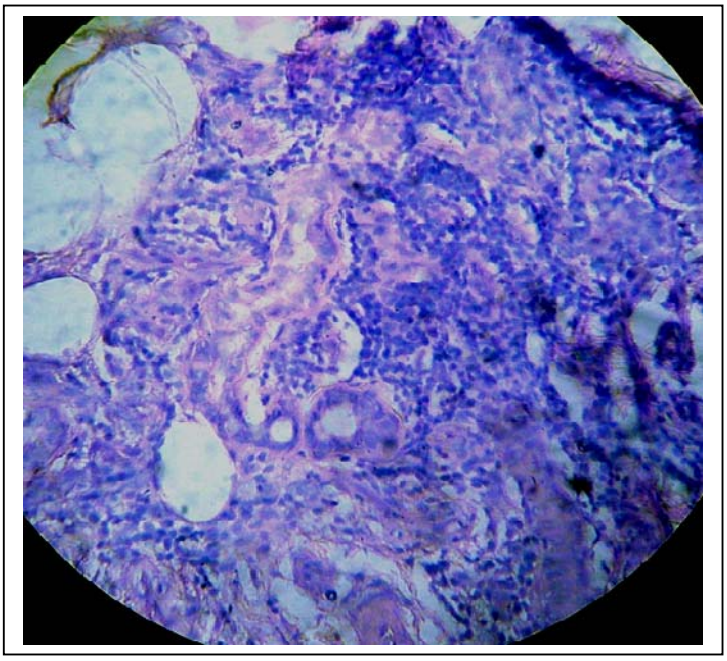
Erythema nodosum showing septal panniculitis with septal widening, predominant lymphohistiocytic infiltrates and granulomas within the septa

ERYTHEMA NODOSUM LEPROSUM



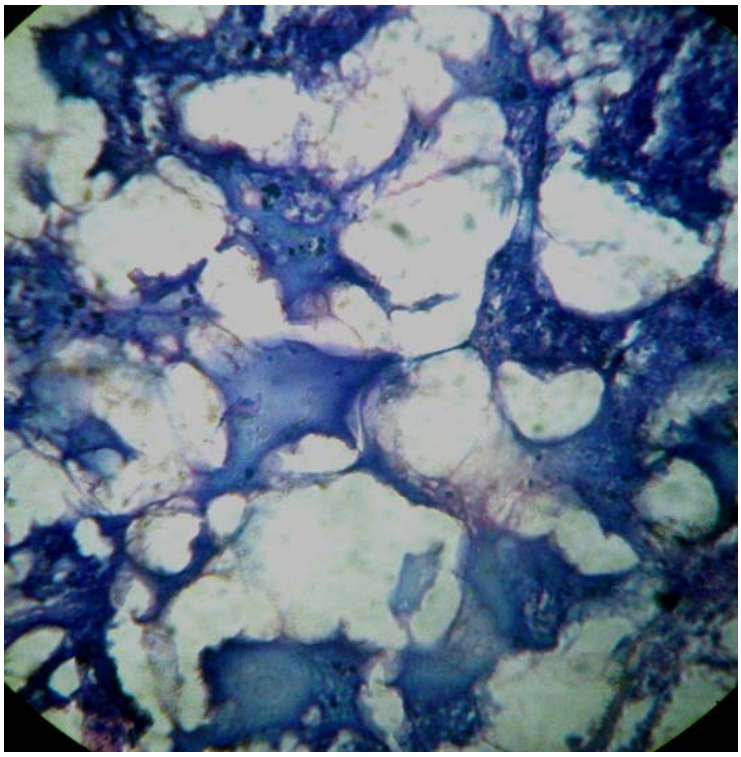
Erythema nodosum leprosum showing lobular Panniculitis with vasculitis

ERYTHEMA INDURATUM



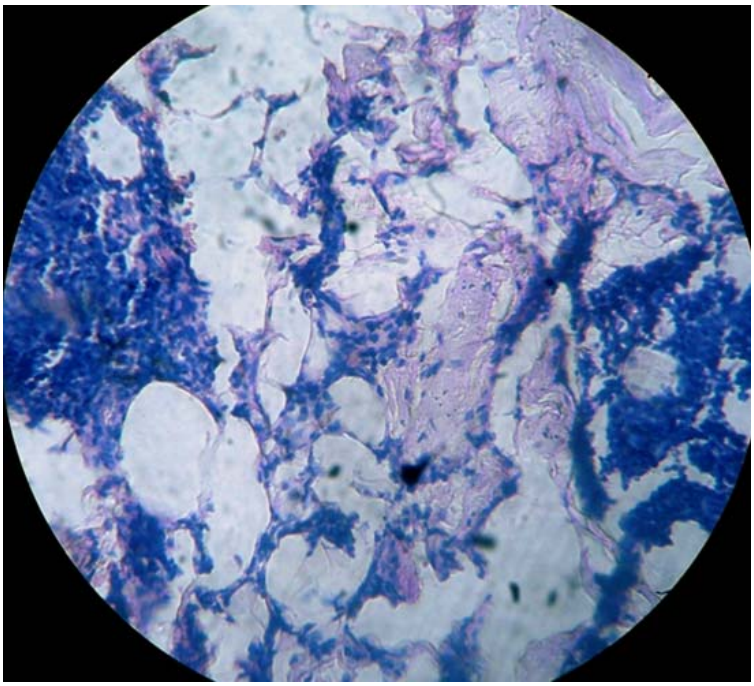
Erythema induratum showing lobular panniculitis with vasculitis, lymphocytic and neutrophilic infiltration and Small epitheloid histiocytes

PANCREATIC PANNICULITIS



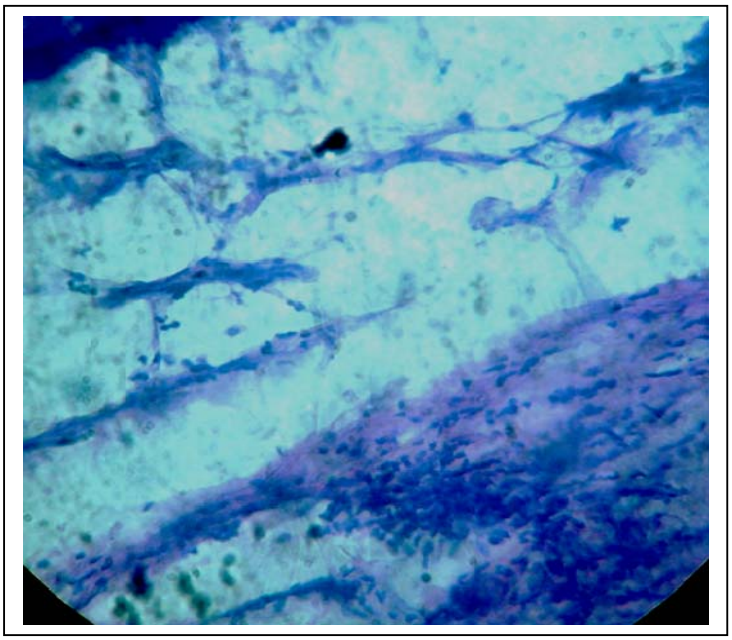
Pancreatic panniculitis showing lobular panniculitis with homogenous basophilic material within the fat lobules

PANNICULITIS OF ARTHROPOD BITE



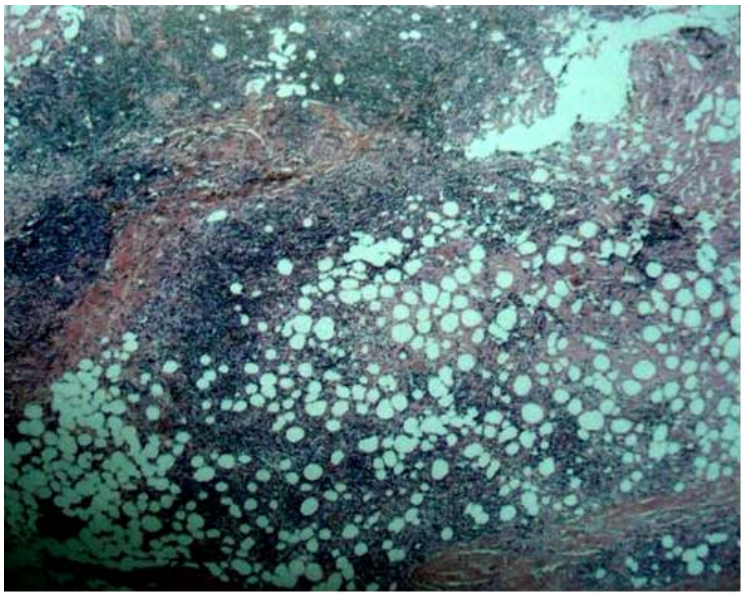
Panniculitis of arthropod bite showing mixed panniculitis with dense lymphocytic infiltration

EOSINOPHILIC FASCIITIS



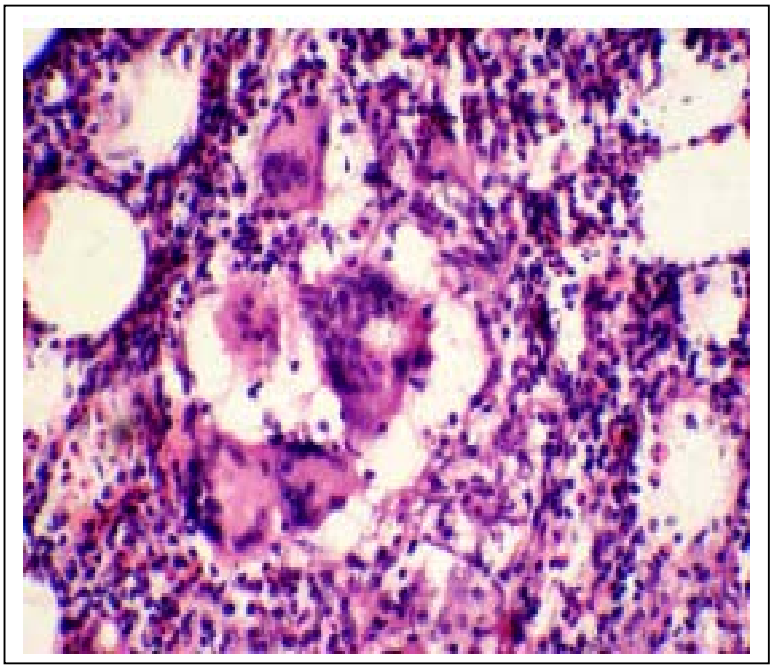
Eosinophilic fasciitis showing septal panniculitis, septal infiltration with lymphocytes, thickening and infiltration of deep fascia

LUPUS PANNICULITIS



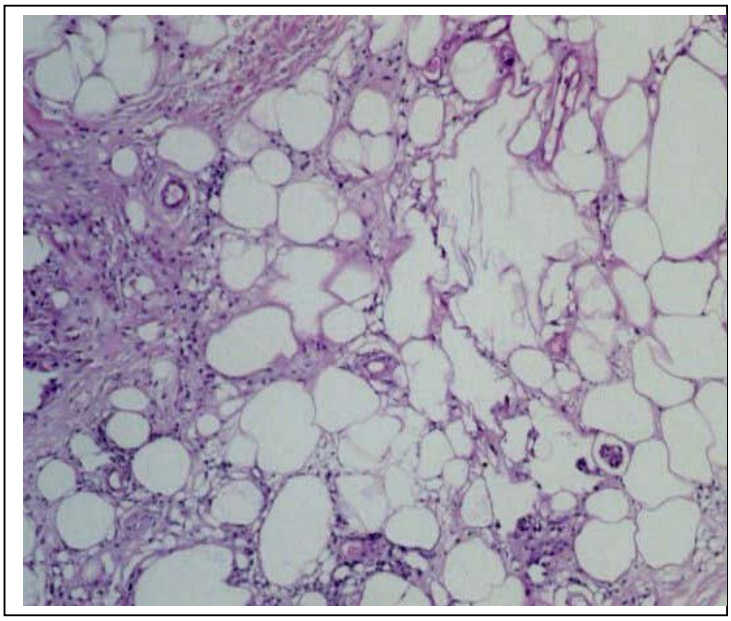
Lupus panniculitis showing a septal and lobular panniculitis with dense lymphocytic infiltration and necrosis of fat lobules.

SUBCUTANEOUS ZYGOMYCOSIS



Subcutaneous zygomycosis showing dense subcutaneous inflammation composed of predominantly lymphocytes, eosinophils and giant cells

LIPODERMATOSCLEROSIS



Lipodermatosclerosis showing mixed panniculitis with septal thickening and mixed cell infiltrate

HIV - ASSOCIATED LIPODYSTROPHY

PROFORMA

Name : Age : Sex :
Hosp.No. : S.l.No. :
Presenting Complaint :
Duration of Skin Lesions
(in weeks)
Type of Skin Lesion : Papule
Plaque
Nodule
Others
Tenderness : Yes/No
Ulceration : Yes/No
Number of Lesions :
Symmetrical : Yes/No
Site : Single/Multiple
Description :
-Scarring : Yes/No
-Others :
Description
Systemic symptoms : Yes/No
Fever : Yes/No
Duration :
Arthralgia : Yes/No
Duration

General Examination
Weight :
Pallor : Yes/No
Temperature :
-LNE : Yes/No
Description :
-Arthritis : Yes/No
Description
-Varicose veins : Yes/No
Description
Systemic Examinations
CVS :
Resp :
CNS :
GIT :

Investigations:

Complete Haemogram
LFT
S. Bilirubin
SGOT
SGPT
Alk phosp
A/G Ratio
Sr. Creatinine
VDRL
HBsAg
Aso titre
HIV
HCV
Skin smear for AFB
ANA
MANTOUX TEST
U/S Doppler

Skin Biopsy

PANNICULITIS	ABSENT	PRESENT
Septal panniculitis		
Lobular panniculitis		
Mixed panniculitis		
Granulomas		
Fat Necrosis		
Vasculitis		
Small Vessel		
Medium Vessel		
Large Vessel		
Epidermal Changes		
Dermal Changes		

Special Stain
1.
2.
3.
Others
1.
2.
3.
Final Diagnosis : clinical Final Diagnosis : Pathological

Clinical spectrum of panniculitis